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MEDICAL SOCIETY

DECEMBER 1963 VOLUME XLI NUMBER 2

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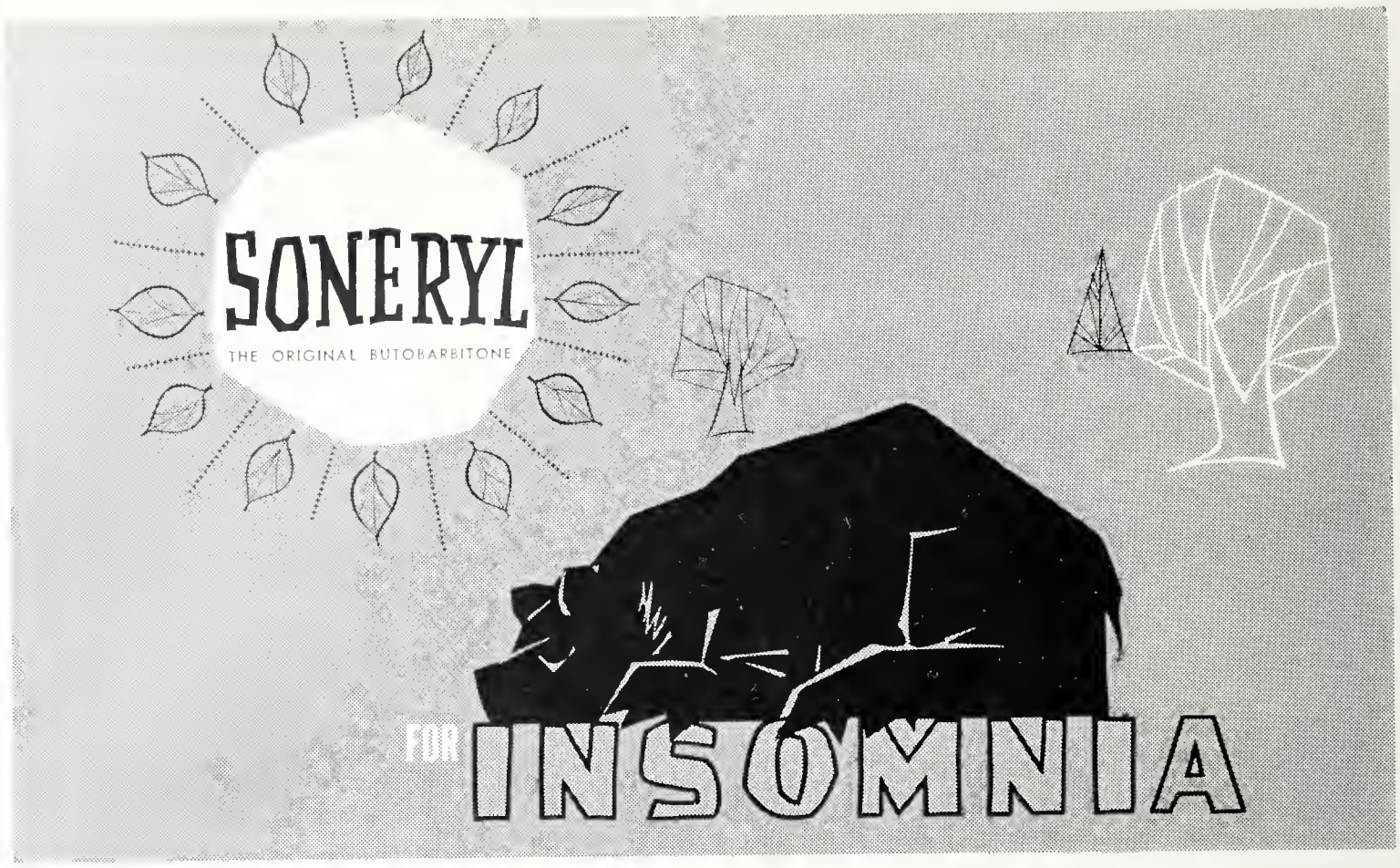

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Editorials

ON BEING IMPERTERBABLE

"In the first plaee, in the physieian or surgeon, no quality takes rank with imperterbability," and I propose for a few minutes to direet your attention to this essential bodily virtue. Imperterbability means coolness, and presenee of mind under all eireumstances, ealmness amid storm, elearness of judgment in moments of grave peril, immobility, impassiveness, or, to use an old and expressive word: phlegm. It is the quality whieh is most appreeiated by the laity, 'though often misunderstood by them, and the physician who has the misfortune to be without it, who betrays indeeision and worry, and who shows that he is flustered and flurried in ordinary emergeneies, loses rapidly the eonfidenee of his patients. As imperterbability largely is a bodily endowment, I regret to say that there are those amongst you who, owing to congenital defects, may never be able to acquire it. Edueation, however, will do mueh and with praetiee and experienee, the majority of you may expeet to attain a fair measure. The first essential is to have your nerves well in hand. I have spoken of this to you on many oecasions and have urged you to educate your nerve eentres so that not the slightest dilator or contraetor influenee shall pass to the vessels of your faee under any professional trial. With sueh advantages, he is so equipped that no eventuality ean disturb the mental equilibrium of the physieian; the possibilities are always manifest and the eourse of aetion elear." (Sir William Oser - Aequanimitas)

Nowhere in medieine is this quality more valuable than in the elinieal emergency. By definition the physieian's cool appraisal of the situation, followed by the institution of rapid treatment is neeessary to prevent irreversible ehange to the patient. For the graduating elass of 6T4, spring holds in store for us the ideal time to demonstrate our fledgling imperterbability. The Dominion Council examiners will be unimpres ed with an abundance of teehnical data if the eandidate is unable to proeeed from basie prineiples and eannot rationally apply his knowledge.

S.W.L.

J. F. K.

The first entered posteriorly and emerged anteriorly after violating the right pleural spaee. He eould have survived exeept that the second shattered the eranium and everything beneath it in the left parietal region. High integrative funetion was eompletely disrupted; life eould not be sustained.

John Fitzgerald Kennedy, father, husband, millionaire, and president of the United States of Ameriea, had been slaughtered.

Eaeh of us, no doubt, shuddered when he heard of the brutal manner in whieh this man had died. This death was so purposeless and violent that even the protective but sick humour of the medieal student was stilled.

The exeeutive responsibilities upon those shoulders elevated him in our eyes. The image projeeted grew god-like before us all. When he was saerifieed and the image shattered, we suddenly and belatedly realized our blasphemy for having diefied a mortal.

His faee has now passed from magazine eovers, his initials are gone from headlines, and soon his name will eease to eross the public's lips. However, men of high purpose and intelligence, publie eoneience, and true aecomplishment will long remember him. For them, he quiekened aspirations into reality and began to make dreams eome true.

Wm. S.C.

On Inspection

by C. Listgarten *Photos by A. S. Mialowsky St.M. Hosp.*

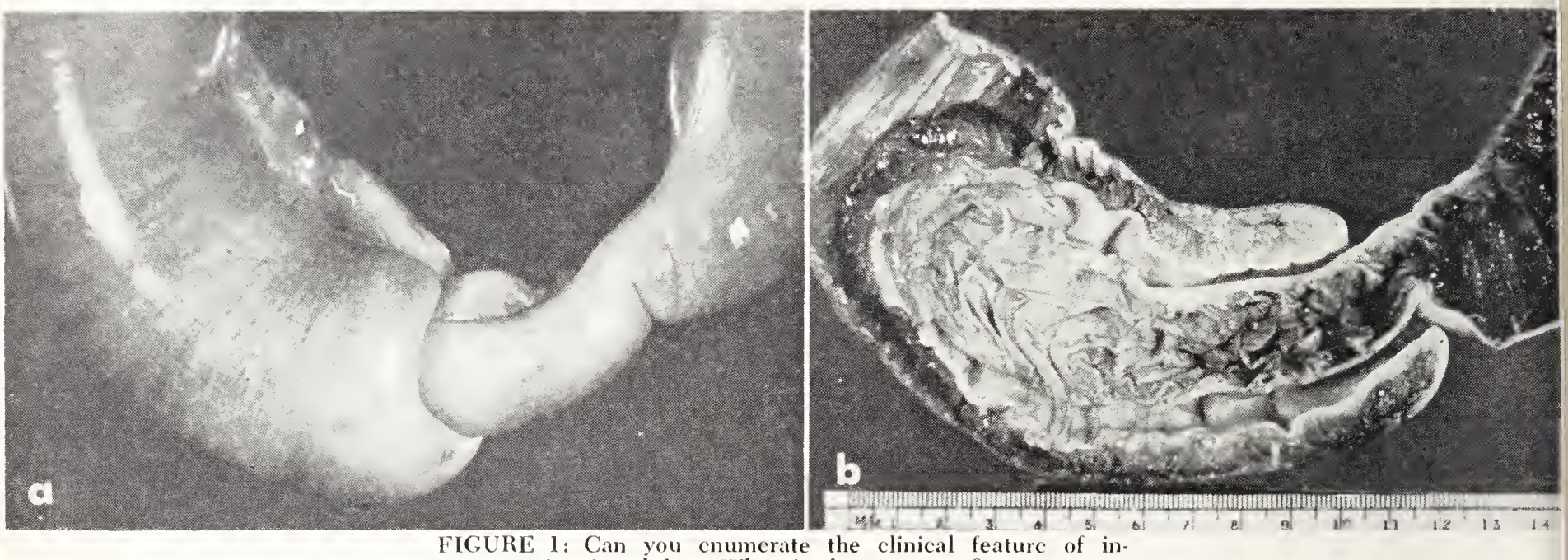

FIGURE 1: Can you enumerate the clinical feature of intussusception in adults. What is the treatment?

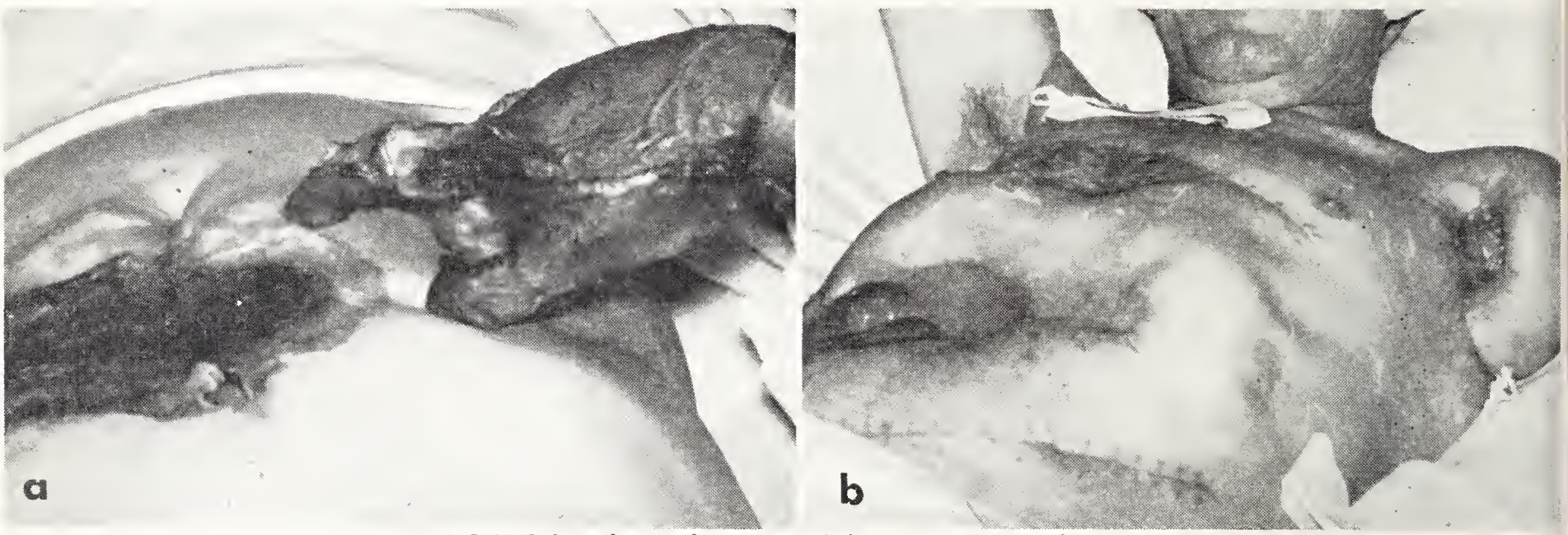

FIGURE 2: What is the nature of the injury illustrated. Do you know its characteristic aspects?

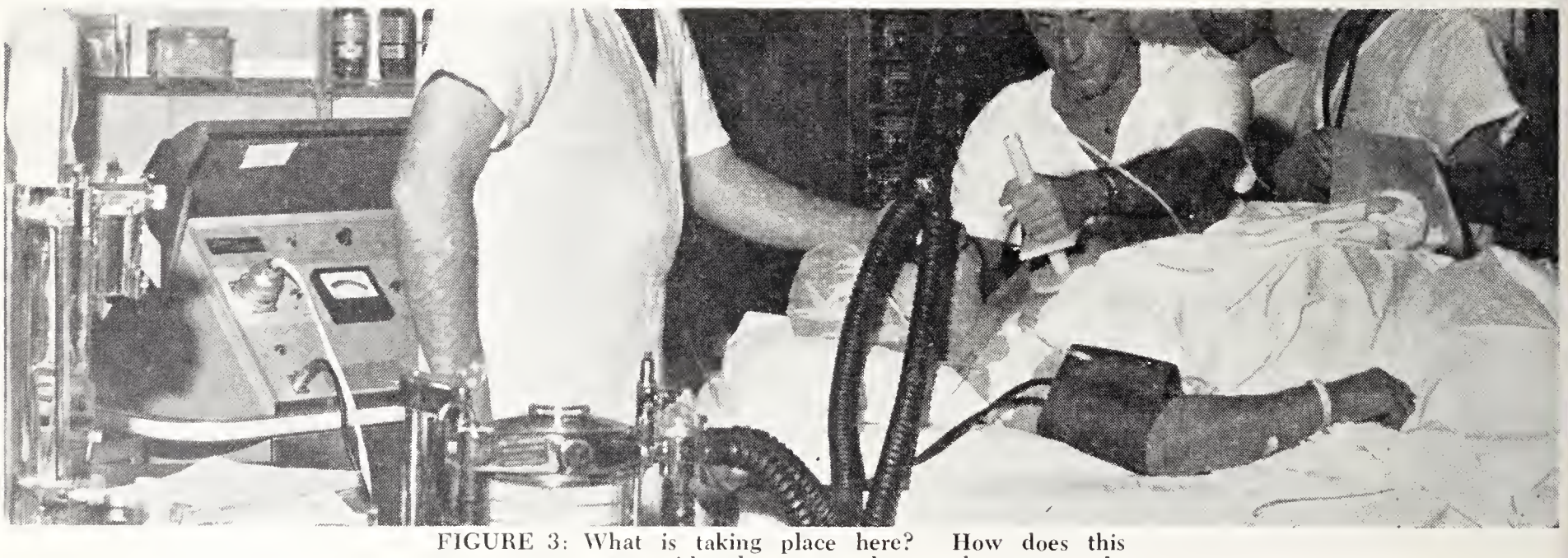

FIGURE 3: What is taking place here? How does this treatment compare with other approaches to the same problem?

The Immediate Management of Closed Head Injuries

D. G. Cockburn 6T4

Close head injuries are one of the commonest and at times the most urgent problems a physician has to deal with. Their immediate threat to life is well known and doctors called upon to deal with them often do so with considerable trepidation. It is ignorance of the basic principles of pathology and treatment that is responsible for this wholly undeserved deputation. In this paper I will outline the pathogenesis of these injuries and then present an outline of treatment based on this pathogenesis. The care that a person with brain damage receives immediately following the injury is the governing factor in whether he recovers or succumbs. Later treatment will to a certain degree determine the extent of complications — such as, brain infection (meningitis or abscess), post-traumatic epilepsy and paresis.

Pathogenesis

By definition, *closed head injury* indicates there is no direct communication between the exterior and the brain substance although there may be scalp lacerations and/or skull fractures. Nomenclature of the types of head injuries tends to be somewhat confusing partly because the clinically descriptive terms are often not accompanied by characteristic pathological changes. I will divide head injuries into the following three types.

1. Concussions
2. Disruption of Brain Substance
3. Cerebral Compression

All head injuries which are accompanied by a period of unconsciousness may be considered to involve at least *cerebral concussion*. The forces transmitted to the brain by the initial injury, whether it be a blow with a blunt object or a laceration with a bullet, radiate to all parts of the brain. These forces account for the initial unconsciousness. The exact means by which these forces exert this effect is not clearly understood although certain recent studies have advanced some plausible hypotheses. It has been shown that following the concussive force the small arteries to mid-brain may go into spasm thereby depriving the reticular substance of the upper brain stem, of its blood supply. In addition the nerve cells including those of the reticular substance undergo a temporary paralysis of function as demonstrated by their loss of Nissl substance. Disruption of brain substance cannot account for the unconscious because commonly parts of the brain are excised at operation under local anesthesia with no loss in consciousness. Under the term *disruption of brain substance* I have included contusion which is brain damage secondary to rupture of small cerebral blood vessels and the laceration which occurs when the brain is torn as with a penetrating injury. The difference between these two is really one of degree and usually one is accompanied by a certain amount of the other. *Compression of the brain* occurs following the formation of surface clots (extradural or subdural) and in cerebral edema which to a certain degree accompanies all head injuries.

Closed head injuries are caused by acceleration — deceleration forces. Acceleration occurs when the motionless head is suddenly put into motion as with a blow by a blunt object. Deceleration occurs when the moving head is suddenly brought to a standstill as occurs in automobile accidents when the head strikes the dashboard or as occurs in falls where a person lands on his head. The more rapid the acceleration (or deceleration) the greater the damage to the brain. The brain because of its inertia lags behind when the head is set in motion, then strikes forcibly against the opposite side of the calvarium and bounces back. The greatest compressive force is thus located on the opposite side of the head to the application of force (contre-coup). Depending on the extent of the force this may cause only concussion or it may cause ecchymoses or disruption of the cerebral tissue. Common sites are the tip of the temporal lobes and the under surface of the frontal lobes. In addition because the head is anchored at the neck and because the force is usually applied tangentially there is usually considerable rotatory force with its accompanying shearing effects. These injuries are followed by unconsciousness of longer duration than in concussion and with a greater degree of total amnesia. The patient may be comatose or semi-comatose for a long period. Depending on the location of the injury localizing neurological signs such as hemiparesis and aphasia may subsequently develope.

The deteriorating level of consciousness occuring subsequent to head injuries is usually due to increasing intracranial pressure. This may be the result of cerebral edema or surface clots. The brain like any other tis-

sue swells with edema when it is damaged. Since the brain is enclosed in a rigid framework a point is reached where further edema is accompanied by increased intracranial pressure and this causes compression and ischemia of brain tissue depressing cerebral function. The increased intra-cranial pressure whether due to edema or hematoma causes herniation of the hippocampus through the hiatus tentorii. (coning.) This herniation may have several consequences, the most important of which is compression of the midbrain with its reticular activating substance. Activity of this reticular substance of the brain stem is postulated to be the mechanism by which cortical activity is maintained. i.e. the conscious state. It receives afferent fibres from all parts of the body and its efferents stimulate the activity of the cerebral cortex. Depression of reticular function leads to subsequent deterioration in the level of consciousness. Hippocampal herniation may in addition displace the midbrain caudally causing stretching and finally tearing of the small nutrient arteries to the midbrain. These midbrain hemorrhages contribute to the damage to the reticular substance. Herniation also causes compression of the oculomotor nerve resulting in a fixed dilated pupil and compression of the posterior cerebral artery with consequent occipital infarction and blindness. It may also compress the cerebral peduncle against the edge of the tentorium cerebelli on the opposite side leading to ipselateral hemiplegia. Hippocampal herniation can be corrected but the brain stem hemorrhages that result from unrelieved herniation are irreversible.

Epidural hemorrhage results from tears of the middle meningeal artery or its anterior and posterior branches. Xrays usually, though not always, reveal a fracture crossing the vascular grooves. Bleeding may also occur from the small nutrient arteries which run from the endostal layer of the dura to the overlying bone or from the dural venous sinuses. If bleeding is severe it will strip the dura from the bone and thus open up a new bleeding sources. The clot of extradural hematoma is always solid and it will cause death unless relieved.

Subdural hematoma usually results from rupture of the bridging veins which run from the surface of the brain across the subdural space to the dural venous sinuses. The veins running up in the mid line are the ones usually involved. Often it is accompanied by extensive underlying brain damage and thus the prognosis is always grave. Acute subdural hematomas are often on the opposite side to the blow and there may be an extra dural hematoma on the same side. Subdural hematomas are usually liquid and very extensive even if they are not very thick.

If the initial subdural hematoma is unsuspected after a few weeks a membrane will form around the hematoma which has semi-permeable properties. Osmotic pressure increases in the hematoma following the breakdown of hemoglobin and water passes into the hematoma causing it to gradually increase in size. This creates symptoms of gradually increasing intracranial pressure.

Features of Brain Injuries

(1) *Depression of Level of Consciousness*

This is the most important sign to watch for after head injury. An estimation of the level of consciousness should be made after every head injury. This should include the patients ability to converse in an intelligent manner and his powers of reasoning, calculating, remembering and comprehension. As the level deepens his response to commands, presence of absurd speech and aimless movement, incontinence of urine and finally his response to physical stimulation e.g. pin prick should be observed and recorded.

(2) *Blood Pressure and Pulse*

In the case of rapidly expanding intracranial lesions the pulse rate falls and the blood pressure rises. This is due to pressure on the brain stem that stimulate vagal and vasomotor centres. Next to the level of consciousness alterations in blood pressure and pulse are the most reliable indicators of sudden increase in intracranial pressure. However these changes will not occur if brain stem centres are damaged by the original injury or subsequent hippocampal herniation.

The following signs occur relatively late and usually indicate irreparable brain damage.

(3) *Dilatation of Pupil*

Pupil dilatation on the same side as the lesion is usually due to distortion of the oculomotor nerve by hippocampal herniation. This usually occurs in the terminal stages. Bilateral fixed dilated pupils are sometimes due to hemorrhage into the midbrain.

(4) *Hemiplegia*

Subdural and extradural hematomas usually spread over an extensive area of the brain distributing their compressing force over the whole hemisphere and therefore the hematoma may be quite large before localizing signs such as hemiplegia occur.

(5) *Loss of Brain Stem Reflexes*

Loss of swallowing and cough reflexes indicate serious depression of brain stem function and contribute

to the respiratory obstruction. Corneal reflex is centred in the brain stem and paralysis of this reflex is a late sign of brain stem damage.

(6) *Extensor Spasm and Periodic Breathing*

Presence of extensor spasm following stimulation and periodic breathing usually indicate irreversible brain stem damage.

(7) *Hyperpyrexia*

In persons whose thermal control may be deranged, the temperature may be very high.

Management of Head Injuries

(a) *First Aid*

Death directly following head injuries is due to either extensive cerebral disintegration for which nothing could have been done or it is due to suffocation for which much could have been done. A few simple measures preformed at the scene of the accident can do more to insure the patient's recovery than innumerable more heroic measures later in the hospital. Three factors accelerate the raised intracranial pressure which develops following the injury.

(1) Increase in cerebral venous pressure secondary to increased intrathoracic venous pressure in obstructed airway (forced expiration), tension pneumothorax and straining from any cause.

(2) Accumulation of CO_2 in the blood due to inadequate pulmonary ventilation causing cerebral edema.

(3) Compression of the neck veins by tight clothing or a bandage around the neck.

Therefore the first aid measures should consist of two important procedures—

(1) Obtain an adequate airway. This includes removal of foreign bodies, dental plates and secretions from the throat, pulling the tongue forward, and putting the patient in a semi prone position.

(2) Loosen the collar to prevent venous engorgement.

To these may be added securing hemostasis in any scalp lacerations. Since the scalp is highly vascular hemorrhage from this source can lead to hypovolemic shock. Bleeding can be arrested in an emergency by firmly compressing the scalp against the skull.

(b) *Immediate Management in Hospital*

(1) The prime concern in hospital should be securing and maintaining an adequate airway. Placing the patient in the semi-prone position, pulling the tongue forward and suction of the pharynx should be accomplished immediately. If this does not assure an adequate airway an endotrachel tube should be passed. If this proves difficult or impossible tracheotomy should be immediately preformed.

(2) The vital signs (level of consciousness, blood pressure, pulse rate respirations and temperature) should now be noted. These must be observed and recorded frequently during the acute stage.

(3) A careful search should be made for other injuries; especially if signs of shock (falling B.P. & rising pulse) are present. Shock rarely follows head injuries except in presence of large scalp lacerations. If shock is present and there are no obvious sources of external hemorrhage, a careful examination of abdomen for rupture of spleen or liver should be undertaken. Injuries to the spine, chest or abdomen may be more urgent than the head injury and should be treated first.

(4) The scalp wound, if present, should now be dealt with, according to basic surgical principles. The wound must be thoroughly cleansed and any devitalized tissue excised. The skull should be carefully examined for signs of fracture both by direct vision and by palpation with the finger but never with a probe for it may push infection through the fracture into the cranium. Every attempt must be made to preserve the blood supply of the skin although owing to the free anastomosis in the scalp a portion almost completely detached may yet survive. Local antibiotics such as Bacitracin should be instilled into the wound but systemic antibiotics usually are not necessary. Tetanus toxoid or antitetanus serum should be given as for any possibly contaminated wound. The scalp should be closed by deep sutures through its whole thickness. If the wound edges cannot be brought into apposition without tension a skin flap should be swung over to fill the defect or a relaxing incision made.

(5) Burr holes should be made immediately if there is a deteriorating level of consciousness that appears to be due to a surface clot. Surface clots are sometimes difficult to distinguish from progressive cerebral edema. The skull films are helpful but sometimes even in the presence of a clot no fracture can be seen crossing a vascular groove. The only way of discovering the cause of the deteriorating level of consciousness is to do exploratory burr holes. Four holes should be made, one in each temporal region and one in each post-auricular region. When the holes are made the dura should be incised to check for subdural hematoma. The procedure is not particularily difficult or hazardous if done carefully in a good light. General anesthesia is preferable for the procedure but it requires an experienced

anesthetist. It may be the only way to eliminate the restlessness of the patient and also if an endotracheal tube is used it prevents the aspiration of blood or secretions while the patient is on his back for the procedure.

(6) Restlessness may interfere with the management of the patient. The cause (distended bladder, painful fracture or tight bandage) should be dealt with. Sedatives are best avoided until the patient has recovered consciousness. Morphine and codeine are always contra-indicated as they depress respiratory function and interfere with pupillary reactions.

(7) Lumbar puncture should never be used in the presence of raised intracranial pressure (papilledema) unless there is a question of meningitis and then it should be preformed with utmost care for it may precipitate or aggravate hippocampal herniation.

Prognosis

An accurate prognosis cannot be given during the acute stage until consciousness is regained and maintained. Extensor spasms, periodic breathing, and lack of arousal to painful stimulation often indicate irreversible brain damage but the ability for functional recovery of the brain is remarkable and consciousness may be regained after several weeks or months. Recovery is a continuing process which may take months or years but once it stops it is not resumed.

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The Diagnosis and Treatment of Convulsive Disorders

A. L. Morrison 6T4

Convulsive disorders are involuntary general paroxysms of muscle contractions of such a dramatic nature that they require the physician to take immediate measures to terminate the seizure and to determine the cause in order to treat them adequately. At the bedside, both therapy and diagnosis will be conducted simultaneously; however, in order to facilitate the discussion, the latter will be considered first.

Diagnosis:

To classify convulsive disorders because of our lack of knowledge of their etiology is a difficult task. For this reason, the anatomic classification seems to be the most useful.

A seizure is caused by an abnormal discharge of impulses from the cerebral ganglion cells, arranged in two areas of grey-matter: the cerebral cortex and the central grey mass. Consequently, seizures may be immediately divided into i)*Cortical* and ii) *Central* or *Centrencephalic* seizures.

Factors outside the cranium also play a part by changing the environment of the brain beyond a certain critical level or threashold, and thereby facilitating the occurence of a convulsion. The third category is then,

Threshold seizures.

Cortical

Cortical seizures are usually due to macroscopic cerebral disease of which the most common is cerebral vascular lesions. Other causes in decreasing order of frequency are: intracranial mass lesions, vascular anomalies, post-traumatic lesions, congenital lesions, inflammatory diseases, and degenerative and demyelinating diseases.

In order to make this diagnosis, we must first establish the fact that the convulsion began in a localized area of the cortex. This is done by taking a careful history of the attack both from the patient and an observer, by noting evidence of cortical disfunction, such as hemiparesis on examination, and by E.C.G. studies which usually uncover 90% of focal abnormalities.

The signs of cortical seizures may vary with the location of the lesion as illustrated in the following list:

Premotor frontal area:

head and eye turning to opposite sides.

Motor strip:

tonic and/or clonic movement in face and/or limbs of opposite sides, sometimes progressing by a Jacksonian march from distal to proximal limb musculature.

Sensory strip and adjacent regions of parietal lobe:

loss of sensations or tingling in the corresponding body part of the opposite side. These may also exhibit a Jacksonian march.

Lateral surface of temporal lobe:

formed visual and auditory hallucinations, which may recreate previous authentic sensory experiences.

Inferior and medial aspects of temporal lobe:

olfactory and gustatory hallucinations.

Occipital lobe:

unformed visual hallucinations or, less frequently, blindness, usually in the centralateral homonymous visual field.

As the abnormal impulses migrate across the substance of the brain, from the initial focus, all certain specificity for localization disappears and a grand mal type of seizure occurs with its generalized tonic and clonic movements.

Centrencephalic Seizures

The reticular substance, that part of the brain which is responsible for our alertness, consists of a mass of central nerve cells which stretch from the thalamus to the upper part of the thoracic cord. It is thought that centrencephalic seizures probably begin in this area; consequently, disturbances in consciousness are initial events in these attacks.

Clinically, there are two types of centrencephalic seizures: grand mal, and petit mal. The former consists of tonic and clonic phases, a stage of relaxation and recovery and a stage of sleep and drowsiness. There may be an aura, which is usually a nebulous mid-line sensation, either in the epigastrium or cranium. All cases have post-ictal confusion.

The latter, petit mal, consists of three clinical types. When they are found together, they are known as the petit mal triad. The first of these is *staring spells,* consisting of short periods of unconsciousness lasting from one to thirty seconds, in which the patient stops his activities and appears blank to the observer. He then resumes his activities unaware that he has had an attack.

Secondly, we may find *akinetic seizures,* lasting less than one second, in which the child falls suddenly to the ground with loss of muscle tone. The third discrete sign is *myoclonic jerking,* consisting of bilaterally symmetrical brief jerks of head of limbs.

The diagnosis in all cases of centrencephalic lesions rests on the fact that there is instantanious loss of consciousness and that the attack is symmetrical. Moreover, there are bilaterally synchronous abnormalities on examination with the E.E.G. and in the case of petit mal, there is a diagnostic three cycle per second spike and wave pattern.

Threshold Seizures

Threshold seizures are secondary to systemic disease as was explained above. A list of some of the more important causes follows:

Metabolic	a) *tetany*: Vitamin D deficiency, hypoparathyroidism and alkalosis.
	b) *hypoglycemia*: idiopathic spontataneous hypoglycemia, adrenal insuffiency, hypopituitarism, hyperinsulinism (overdose), tumour of islet cells of Langerhans, Galactosemia, hypothyroidism.
	c) *Vitamin B insufficiency,* Pyridoxine deficiency or dependency.
Infection:	febrile convulsions, sepsis, viral
Toxic:	Acute lead poisoning, Atropine, Boric acid, Aminophylline, Salicylates
Cerebral Anoxia:	secondary to aspiration, heart block, syncope, shock, blood loss
Emotional	Hysteria, breath holding

The diagnosis of this type of seizure is made by i) taking a complete history noting such points as birth trauma, family history, pica, drugs, fever and description of convulsions, ii) a thorough physical examination, iii) special investigation such as X-Ray of chest and skull, urinalysis, F.B.S., N.P.N., Serum calcium, examination of C.S.F., and an E.E.G.

In order to make the diagnosis of a febrile convulsion, Prichard suggests that you must have the following criteria:

1. patient hitherto neurologically normal
2. age between six months and four years
3. temperature of 100 degrees F. or greater
4. symmetrical seizure
5. seizure lasting less than thirty seconds
6. an apparently normal child after the seizure
7. a normal E.E.G. when the fever is gone

TREATMENT

General Treatment

The main aim of treatment during a convulsive seizure is to prevent personal injury. A wadded end of a handkerchief, a gauze roll, a well-padded tongue depressor, or a folded leather belt should be placed between the teeth to prevent tongue biting. Tight clothing, especially collars, should be loosened and the patient's head should be elevated on a pillow or folded coat. Attempts at restraint should be limited to preventing the patient from falling out of bed or rolling into furniture. The best way to prevent the former is for two assistants to place their bodies across the patient's chest and knees, and take a good hold of the opposite side of the bed.

Since most seizures stop spontaneously in less than a half-hour, sedatives or anaesthetic drugs are not indicated unless the seizures exceed this time limit or tend to occur in a series. In these instances, any of the volatile anaesthetics may be given as for example; ether by mask. At the same time, make sure that the airway is clear and administer oxygen if available. Intravenous barbituates or paraldehyde are more often used because of their convenience and greater effectiveness. In adults, intravenous sodium phenobarbital O.3G. to O.5G. or paraldehyde 3 c.c. to 6 c.c. are used. In children, give intramuscular sodium phenobarbital 2 to 3 mg. per pound body weight of I.M. paraldehyde 1 c.c. per 10 pounds body weight. Half this amount may be repeated in twenty minutes if necessary.

Specific Therapy

Cortical Seizures:

The treatment of cortical seizures depends on whether the causative agent is progressive or not. If it is progressive such as a tumour, an abcess or a subdural haematoma, then there must be surgical intervention. However, if its is not progressive, such as a patch of gliosis, the seizures should be controlled with the smallest amount possible of the least harmful drug. The choice of drugs is essentially the same as that for grand mal convulsive disorders and will be discussed under that heading.

Centrencephalic Seizures:

The treatment of centrencephalic seizures is medical. Some of the drugs commonly used are:

Grand Mal

(1) Sodium Phenobarbital:—This drug has a high theraputic index and a low toxicity. It is used mainly in patients with frequent grand mal seizures (from one to four per year). Initial dosage is usually 30 mg. T.I.D. and increased slowly to as much as 0.3 G. per day if necessary. Side effects are drowsiness, mental dullness, nystagmus, staggering and skin eruption. Only the latter necessitates discontinuing its further use. The other side effects are controlled by reducing the dosage.

(2) Diphenylhydantion (Dilantin):—Is the drug of choice when seizures are more frequent than four per year. The initial dosage is 0.1 G. b.i.d. and may be increased to a total of 0.6 G. per day. The side effects consist of ataxia of gait, hypertrophy of gums, nystagmus, and in larger doses, stupor or coma.

If the above two drugs are used in combination, a full therapeutic dose of each drug must be given.

(3) Primidone (mysoline):—May be used instead of diphenylhydantoin in cases of severe gun hypertrophy. The dosage is 0.125 G. b.i.d. and its toxic effects are giddiness, vomiting and ataxia.

Other drugs rarely used are Mesantoin, which has the serious toxic effect of agranulocytosis, and bromides.

Petit Mal

At the onset of petit mal seizures one of the best drugs to try is acetazolamide (Diamox) in doses of 0.25 to 0.75 G. per day. It is of particular use in staring spells that are initiated by hyperventilation as it produces mild ketosis. If after one week of therapeutic trial there is not a great reduction in the number of attacks, other drugs should be resorted to.

Trimethadione and paramethadione are other drugs of choice. They are given in doses of 0.9 to 1.5 G. daily to children over six years of age, and doses of 0.3 to infants. Toxic manifestation are skin eruptions, photophobia, and aplastic anemia. Because of the latter complication, monthly blood counts during the first year of use are recommended.

Methyl phenylsuccinamide (Milontin) is an effective drug for petit mal seizures. The adult dose is 0.9 to 3.0 G. per day while the dose for children is 0.6 to 1.8 G. per day. It is less toxic than the others but occasionally may produce reversible albuminuria and haematuria.

Threshold Seizures

In treating thresold seizures, the main aim is to treat the systemic disease or disorder which is causing the convulsion. In the case of febrile convulsions, one would first attempt to lower the temperature of the patient by antipyretics such as aspirin and by sponge baths. Secondly, one would administer antibiotics to eliminate the infection.

If the patient is in a hypoglycemic state, he should be given I.V. glucose 50% in 50 c.c. of water, A positive response may occur in minutes.

If the patient has tetany due to hypocalcemia he should be treated with 10 to 20 c.c. of 10% calcium gluconate I.V. at the rate of one to two c.c. per minute. This may be repeated in one hour if necessary.

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Medical Horizons

"Wonderful Wonderful Copenhagen"

Sometimes I get the impression that I know some of the cities in Europe where I spent only 3 or 4 days better than I know Copenhagen where I stayed a month. But then I remember that I spent very little time in Copenhagen's museums, historical sites or tourist traps. I tried to live like a Dane and so instead of remembering what you'll see on postcards, I remember people — on the subway or bus, on the streets as I passed them on my bicycle, in the hospital, along the canals and parks on an afternoon's walk through the city.

There is no doubt in my mind that it is the Danish people that make Copenhagen a wonderful city, not only to visit, but to live in. They have this reputation all over Europe and their friendliness and hospitality was demonstrated the first day I arrived. I was guided by some strangers to the office of the International Medical Cooperation Committe and the exchange officer promply drove me to his home where his mother already had some beer waiting for us. — Tuborg Gold Cap (nothing but the best!). In the next four weeks I spent a good many hours on their sunporch discussing Hemingway, drinking brandy, and admiring the wonderful Danish early summer. It was through these hospitable people and a fourth year Medical student I became friendly with that I came to know something about Danish people. They have a calm and sensible attitude toward life and enjoy it as much as possible without running themselves ragged making money and keeping up with the Nielsons. Also, they treat everyone alike, no matter whether you are visitor or have lived in Copenhagen all your life. And believe me, this friendliness is infectious. Copenhagen was the only city in Europe where being a tourist doesn't label you as a sitting duck for every crook, short-change artist, and conman out to get his greasy fingers on the almighty Yankee dollar.

My externship in Medical Department B of Bispebjerg Hospital on the outskirts of the city lasted four weeks and the most important thing that I learned can be summarized in one sentence — the world of medicine does not revolve about the University of Toronto or the Toronto General Hospital. This may at first seem to be a naive truism, until you think about your medical knowledge and realize that all you know is that set of practices, therapeutic and diagnostic procedures, and etiological viewpoints drummed in by professors and clinicians of our university and our hospitals.

I'd been there only a short time when I noticed that when there was a question of renal functional impairment a serum creatinine determination was done. When I asked whether the patient's BUN had been measured, the professor tolerantly informed me that BUNs had not been done for 10 years because they were so unreliable. My Toronto-taught arguments of the disadvantages of serum creatinines didn't make much impression on him. Another difference was their routine use of Lactic Acid Dehydrogenase level in any patient either proven or suspected of having a myocardial infarction for diagnosis and indication. I can vaguely remember having been told in IInd year that we sometimes use it.

I was quite surprised by their routine use of chlorpromazine for day and night sedation, having been intimidated by our teachers as to its serious toxic effects on the bone marrow and liver. The professor however calmly informed me that he found it quite useful and had not had much trouble with it at all. Even cirrhotics were taking it. This made me think twice about the unquestionable and universal verity of our teaching and I resolved to use it whenever I thought it necessary. The examination of cirrhotics proved to be quite interesting in that it showed how a socialized state can go too far in its aims. The professor had presented on rounds an alcoholic with cirrhosis, and then remarked to me that any person who could demonstrate that he drank enough to prevent him from working could apply for and receive an Alcoholics Pension. He said that he realized it sounded odd but at least alcoholics did not go hungry. I added "or thirsty either".

My investigation into their system of socialized medicine was not a detailed one but I did make a few observations. People received good medical care and did not have to worry about how they would get or pay for it. Hospitals had adequate modern equipment and

(see page 84)

Cardiac Emergencies

J. R. Newall 6T4

Webster's dictionary defines an emergency as any unforeseen combination of circumstances which necessitates immediate action. However, even in the haste dictated by a cardiac arrest, one should not abandon the principles of good medicine which include logical thinking and reflection upon freshly acquired data. The emphasis in the following discussion will be primarily on myocardial infarction or cardiac resuscitation inasmuch as it represents the most dramatic approach to the leading cause of death today.

Physiology

A state of cardiac emergency may be declared in any condition that causes impairment of cardiac output severe enough to threaten life. Cardiac output is defined as being equal to the product of stroke volume and heart rate. Any cardiac condition seriously interfering with stroke volume, heart rate, or both, could give rise to a crisis calling for immediate action. In heart rates greater than 120 per minute, the stroke volume begins to decrease so that although cardiac output may increase, it does so with decreasing efficiency. At rates over 160, the cardiac output may fall precipitously. Furthermore, coronary blood flow is decreased as diastole is shortened since it is during this period that blood flow through the coronary arteries is maximal. In bradycardias, owing to the long diastolic pause, the diastolic pressure is diminished which in turn tends to decrease coronary blood flow. If the efficiency of the heart is already impaired by ischemic heart disease or any of the myocardopathies, this further reduction in efficiency caused by impaired coronary blood flow could precipitate a dangerous arrhythmia with a possible fatal outcome unless there is quick medical intervention. The latter explains the potentially dangerous situation inherent in ischemic heart disease and arrhythmias.

Conditions which may give rise to acute cardiac failure or cardiac arrest:

1. Ischemic heart disease
   —angina pectoris
   —myocardial infarction
2. Arrhythmias such as
   —paroxysmal atrial tachycardia
   —atrial flutter
   —atrial fibrillation
   —ventricular paroxysmal tachycardia
   —ventricular fibrillation
   —heart block
3. Acute myocardopathies
4. Cardiac tamponade
5. Acute left ventricular failure

Of the preceding conditions, ischemic heart disease is by far the most common and in fact, myocardial infarction is statistically the leading cause of death in North America. It may be argued that this classification is misleading since arrhythmias are not a separate entity but merely sequelae of preceding heart disease such as ischemic heart disease or myocardopathies. This is true; however, for ease of classification, they have been separated under their diagnostic labelling. All of these conditions may progress to a fatal arrhythmia such as ventricular fibrillation or complete asystole, an occurrence which until the last few years was invariably fatal. Now however, new concepts in cardiac resuscitation provide hope in situations which were previously hopeless.

Cardiac Resuscitation

One and a half years ago an intensive care unit for the specific study and treatment of patients with acute myocardial infarcts was organized at the Toronto General Hospital. This "Coronary Unit" provides intensive nursing care, continuous E.C.G. monitoring of the patients, and most important, facilities for dealing with cardiac emergencies and the efficient institution of cardiac resuscitation. In the first year of operation, two people who were "dead" were successfully resuscitated and are still walking around. A somewhat similar arrangement — a joint cardiorespiratory service — was established in Montreal at the Royal Victoria and The Children's Hospital. The diagnosis at the time of resuscitation included not only myocardial infarct but also: respiratory anoxia, uremia, valvular disease, digitalis toxity, Stokes-Adams, miscellaneous and unknown. In this study, a reported 13.5% were successfully resuscitated and survived until after discharge. The important lesson to be gained from both these projects is that cardiac resuscitation can save the lives of persons whose fate would otherwise be sealed.

Procedure

The four "-ates" of successful procedure are:

1. Ventilate

2. Circulate
3. Medicate
4. Defibrillate

To these could be added a fifth principle of equal importance: — post-resuscitation care.

1. *Ventilate*:

Maintenance of a clear airway is of prime importance and for this purpose a rubber mouthpiece will do nicely if available. Assure yourself first, of course, that there is no obstruction present such as false teeth. Next apply a rubber face mask and "bag" the patient with an anesthetic bag. If these pieces of apparatus are unavailable, mouth to mouth respiration is immediately instituted until the proper equipment arrives. With the arrival of competent assistance (presumably notified at the time of cardiac arrest) the patient may be intubated and respiration carried on by bagging the patient.

2. *Circulate*:

Meanwhile, no time must be lost in the institution of external cardiac massage since interruption of cerebral circulation for periods greater than three to four minutes produces irreversible brain damage. Kouwenhoven et al (1960) clearly demonstrated that by rhythmic application of pressure on the sternum 60 times a minute, the heart can be compressed between the sternum and the spine so that an adequate cardiac output can be maintained. This is effective in either ventricular asystole or fibrillation for periods up to 1 to 2 hours. For heart compression to be made mechanically feasible, the patient must be on a firm surface. For this, a board (or tray) is placed under the patient's chest. The heel of one hand is placed over the lower third of the chest. Short quick jabs are applied with the other hand against the dorsum of the first hand, thereby depressing the chest one to one and one-half inches at a rate of sixty per minute. Efficient cardiac massage should produce readily palpable femoral pulses. If the patient is being "bagged", three cardiac compressions are followed by one respiration, thus: 1 - 2 - 3 - bag, 1 - 2 - 3 - bag, and so on. If the patient is being respirated by mouth to mouth respirations, twenty cardiac compressions are followed by three respirations.

3. *Medicate*:

Administrate intravenously either (or both) adrenaline, 1 ml. of 1: 1,000 in 10 c.c., or Isuprel 2 m.g. in 200 ml. of glucose and water. Acute acidosis accompanies cessation in the circulation, hence sodium bicarbonate, at least 180 m Eq., should be given i.v. during resuscitation and repeated if the circulation is not restored within a few minutes. Calcium gluconate, 10 ml. of 10% solution, is also given i.v. to not only stimulate the heart, but to counteract the developing hyperkalemia (with cessation of circulation and by action of adrenaline there is an outflowing of potassium ions into the circulation — sod. bicarb also helps to counteract this shift). Corticosteroids may also be administered since in severe shock the adrenals are likewise thrown into shock. Pressor agents such as aramine or levophed help to support blood pressure in the event of commencement of cardiac contractions.

4. *Defibrillate*:

If ECG patterns reveal ventricular fibrillation, the heart is defibrillated using either the AC external defibrillator starting with 250 volts and working up to 750 volts if fibrillation persists (time duration of current — 0.25 secs.) or the DC external defibrillator starting with a current of 80 joules and increasing to 400.

Efficient utilization of the defibrillator demands the prompt setting up and subsequent examination of tracings of an ECG prior to defibrillation.

5. *Post-resuscitation Care*:

It has been found that resuscitated patients are particularly susceptible to multiple cardiac arrhythmias and hence should be closely followed by continuous monitoring. With some patients, it may be necessary to introduce intravenously an electrode catheter so that its tip is in contact with the endocardium of the right ventricle and by this method stimulation may be initiated at a rate sufficient to suppress further arrhythmias or, in fact, act as an internal temporary pacemaker.

Although cardiac resuscitation is a most exciting and dramatic measure, it is to be hoped that the patient is seen long before it is necessary to use such desperate efforts.

1. *Ischemic Heart Disease — Acute Myocardial Infarction.*

As we grow older the coronary arteries become gradually narrowed by arteriosclerotic processes and atheromatous plaques until eventually one may become completely blocked by thrombosis, subintimal hemorrhage into a plaque, or an embolus. Let us assume that you have just responded to the urgent call of a distraught wife whose husband has just suffered a severe coronary artery occlusion. As you enter the room and gaze upon the hapless victim of this "number one killer", the diagnosis becomes almost a certainty. He is stretched out

on the chesterfield in obvious distress, his face is pale and drawn, every line plainly expressing anxiety and apprehension. He is perspiring freely. He rubs his chest with his hands in a vain attempt to ease the agonizing, vice-like, persistent, substernal pain and moves from side to side in a futile endeavour to find a comfortable position. On questioning, he may complain of pain radiating to the left arm or shoulder, or perhaps to the right arm, or even to both sides of the neck or down to the abdomen. What will you do?

Confirm your diagnosis! A rapid but thorough examination may reveal blood pressure of shock level, a thready, perhaps absent radial pulse, weak heart sounds with a rate above 100. Gallop rhythm, atrial fibrillation, atrial and ventricular tachycardia may be present. A friction rub may be the final clue in the diagnosis. Moist râles may be a consequence of the failing heart.

No time should be lost in transporting the patient to a hospital where ECG and lab examination can reinforce your diagnosis. The ECG may show elevation of the ST segment, inversion of the T waves and the presence of Q waves. These changes will be most obvious in chest leads over the affected area of heart muscle, however, they may not appear for as long as several days. Within twelve hours, rising SGOT levels reflect muscle damage. Values of over 300 usually indicate a fatal outcome. There is usually a leukocytosis of from 12,000 to 15,000, possibly higher. ESR becomes elevated within two to four days.

Even before the patient is hospitalized, his pain and apprehension should be relieved by the prompt administration of 15-30 mgm. of morphine sulphate subcutaneously, to be repeated in 30 minutes if pain is not relieved and repeated thereafter every four hours as required. Later in the illness, anxiety may be relieved by phenobarb 15-30 mgm. T.I.D. accompanied by a bedtime sedative to insure a good night's sleep.

Oxygen by nasal catheter, face mask, or oxygen tent if necessary, ensures the failing heart muscle of an adequate supply of this essential gas.

Anticoagulent therapy is now contemplated. It is administered to those with no contraindications to control thromboembolism. The initial dose of dicoumarol is 300 mgm. given preferably the day of the attack. Subsequent doses, usually ranging from 50-100 mg. are governed by the daily prothrombin time which should be maintained between 2-2½ times the control.

Coronary dilators, such as peritrate 20 mg q4h or nitroglycerine 0.45-0.64 mg. sublinqually are strongly advocated by some to alleviate coronary insufficiency.

Ideally, the patient should be put into an intensive care unit such as the "coronary unit", where he can be continually monitored and subsequently treated at the first sign of developing, potentially fatal arrhythmias. It is interesting to note that although two-thirds of the deaths due to myocardial infarction occurred within the first forty-eight hours, only 2% die after the fifteenth day, proof enough that this disease indeed calls for prompt and efficient measures.

2. *The Arrhythmias*

(1) Paroxysmal atrial tachycardia is a rapid regular heart rate, usually about 170 beats per minute or faster, characterized by sudden onset and offset. The attacks may last minutes or days and are likely to be repetitive.

The cause is usually unknown although it may be associated with some form of heart disease or, rarely, with excessive drinking or smoking, acute infections, or thyrotoxicosis.

Although the attack may be associated with palpitation, fullness of neck veins, faintness or even pain, all of which produce anxiety, they are usually short-lived. If the paroxysm is prolonged, however, heart failure may supervene.

The keystone of therapy in anxious patients is proper reassurance coupled with attempts to prevent the seizures. Increased vagal tone caused by carotid pressure, the valsalva manoeuver, or the mechanical induction of nausea may stop an attack. Other individuals may require digitalization or quinidine therapy. Intravenous or subcutaneous neo-synephrine in a 1 mg. dose may be given if digitalization fails and if there are no contra-indictations to its use. It should be emphasized that P.A.T. can cause ECG changes that might be confused and ventricular fibrillation or myocardial infarct.

(2) Atrial fibrillation is a condition in which the atria are ineffectively contracting at rates of 350-600. The ventricles respond erratically to the rapid impulses from the A.V. node and contract irregularly at a rate of 100 to 150 times per minute.

Rheumatic heart disease with valvular lesions, ischemic heart disease and thyrotoxicosis are the usual underlying causes.

Symptoms include palpitation, fullness of neck veins, apprehension, dizziness and even faintness. A pulse deficit is present and increase with exercise. Electrocardiographic examination reveals absence of normal P waves, presence of fibrillation (f) waves, and irregularly spaced QRS complexes.

In serious cases in which the ventricular rate is very rapid and especially if there is underlying heart disease, bed rest is recommended, followed at once by digitalization. In less severely ill patients without serious underlying heart disease and in whom fibrillation has not been present for a long time, quinidine may cause successful conversion. Quinidine may also be tried in the more severe cases but only after digitalization. If conversion attempts are unsuccessful by medical means, electric defibrillation should be tried.

The seriousness of this condition arises from the fact that the ventricular rate may become so rapid, irregular, and ineffective that the heart will go into failure.

(3) Atrial flutter is a condition in which there are very rapid but regular atrial contractions with a rate of 250-350. The ventricles may respond to every other beat (2:1 block) or slower (3:1 or 4:1 blocks).

The etiology and symptomatology are similar to atrial fibrillation. The ECG displays typical "saw-toothed" flutter waves.

Primary digitalization to produce 3:1 or 4:1 block followed by attempts at conversion with quinidine is the accepted treatment.

The danger of atrial flutter lies in the possible development of a 1:1 block producing syncope and rapid heart failure.

(5) Ventricular fibrillation is usually a terminal event in which the normal ventricular contractions are replaced by a rapid irregular twitching.

It may be associated with electrocution, chloroform or cyclopropane anesthetic, myocardial infarction, digitalis or quinidine overdose.

Clinically, the condition is for intensive purposes a CARDIAC ARREST and should be treated at once by the previously described cardiac resuscitation.

(6) Heart block is a delay in or obstruction to impulse conduction through the AV node. It may be simple (a delay in conduction), partial, or complete (a partial or complete obstuction in conduction). The hazard especially important to patients with organic heart disease and partial or complete block is the sudden slowing or failure of the idioventricular rhythm which results in cessation of effective ventricular contraction for several seconds or longer. During this time the patient may become lightheaded, faint, have convulsions, or if the standstill is long enough he may die.

During syncopal seizures, adrenaline, in 1:1000 aqueous solution in dose of 0.2-0.3 ml. subcutaneously or intramuscularly every 20-30 minutes, may be necessary to prevent fatal asytole. Isuprel, 15 mg. q2-6h sublingually, may prevent further such Stokes-Adams attacks.

3. *Acute Myocardopathies*

This group of conditions, frequently associated with viral infections, are perhaps more common than previously thought. The prognosis is usually favourable although in some cases the development of serious arrhythmias and consequent heart failure demand prompt treatment as outlined.

4. *Cardiac Tamponade*

The symptoms of cardiac tamponade are primarily due to the sudden fall in cardiac output and systemic congestive failure. Orthopnea with little or no pulmonary congestion is usually present. An important sign is the readily percussable, enlarged (as much as 5 cm. to the right of midline) right ventricle.

The mechanical interference with the pumping action of the heart caused by the presence within the pericardial sac of the large amounts of exudate, transudate, or blood is relieved by draining the contents of the sac. Pericardial paracentesis is performed at a point just lateral to the apex beat; however, even repeated insertions of the needle may not remove much of the fluid due to adhesions, etc. In this case the only alternative is a surgical approach.

5. *Acute Left Ventricular Failure*

Although this disorder most frequently occurs in the course of hypertensive heart disease, it is also seen in acute myocardial infarction, valvular heart disease, and the administration of large amounts of blood or saline to a patient with heart disease.

Intense dyspnea with marked anxiety and the production of frothy white or blood tinged sputum are the prominent initial symptoms.

Fifteen to 30 mg. of morphine should be given subcutaneously immediately unless pulmonary or cerebral disease is present. One hundred percent oxygen by positive pressure should be administered. Rotating venous tourniquets are applied at the proximal ends of the extremities using blood pressure cuffs set at 45-50 mm. Hg. Intravenous digitalization is accomplished by giving 1.6 mg. Lanatoside C slowly over a period of 3-5 minutes. Aminophylline 0.25 Gm. is given slowly I.V. and Thiomerin 2cc. I.V. subcutaneously, or intramuscularly are valuable adjuncts to the foregoing therapeutic measures.

Summary

The major cardiac emergencies and their treatment are discussed with the emphasis on initial treatment.

(see page 88)

Chest Emergencies

W. L. M. King 6T5

The thorax is a frequent site of severe injury with the commonest cause being automobile accidents. To the physician called to the scene of an accident or attending at the emergency department of a hospital, three signs demand urgent detection and may provide the "tip-off" to emergency status. These are:

(i) dyspnea

(ii) shift of the mediastinum

(iii) systemic hypotension

Dyspnea can be rapidly assessed using the criteria of Grant, Wright and Clifton:[1]

(1) the rapidity of tachnopnoea

(2) the paucity of chest expansion and the extent of inspiratory intercostal indrawing

(3) the dearth of gaseous exchange audible at the mouth

(4) the intensity of cyanosis

(5) the excessive use of an accessory respiratory muscles (to this might be added the other signs of laboured respiration such as flaring of the alae nasae)

(6) the depression of cerebral activity

The presence of severe hypoventilation should spur the physician to immediate therapeutic measures lest permanent damage result from cerebral anoxia.

Shift of the mediastinum and the trachea in the recently injured patient is very suggestive of pneumothorax and/or hemothorax with the considerable reduction in ventilatory capacity which these entail.

Severe systemic hypotension should direct the physician's thinking along the lines of hemorrhagic shock, cardiac tamponade, etc. These are problems requiring immediate attention and are discussed in other articles in this journal.

In the following discussion, four representative conditions will be considered:

(i) Obstruction of the Airway

(ii) "Stove-in-chest"

(iii) "Sucking" chest wounds

(iv) Tension pneumothorax

These conditions have been chosen for their frequency and extreme nature but it should not be construed that these are the only chest injuries which can assume emergency proportions.

I OBSTRUCTION OF THE AIRWAY

Airway obstruction can be one of the most rapidly fatal chest emergencies without swift and decisive treatment. It is particularly prevalent in children who have an enormous capacity for putting anything and everything in their mouths. The list of commonly found foreign bodies is virtually limitless; food, buttons, toys, coins, candy, fruit seeds, false teeth, vomitus, the tongue in unconscious patients and many other objects are seen frequently.

The severity of the distress depends on the size and location of the foreign body. Small objects which become lodged in bronchus or bronchiole will inevitably, if untreated, produce serious lesions such as atelectasis and bronchiectasis, however, they will not produce the severe distress of asphyxia which is characteristic of the clinical emergency caused by tracheal obstruction.

The symptoms and signs of airway obstruction are graphically portrayed by Morse and Colcord.[10] "The patient is seized with a feeling of annihilation, he gasps for breath, looks wildly around him, coughs violently, and almost loses his consciousness. His countenance immediately becomes livid, the eyes protrude from their sockets, the body is contorted in every possible manner, and froth and sometimes even blood issue from the mouth and nose. Sometimes he grasps his throat and utters the most distressing cries. The action of the heart is greatly disturbed and not infrequently the individual falls down in a state of insensibility, unable to execute a single voluntary action. In short, he is like one who has been choked by the hand or by the rope of the executioner." Where the patient is found unconscious with no history to aid in the diagnosis, the most important features are the profound cyanosis and the distorted facies. Foreign bodies which do not completely occlude the trachea will produce "a palpable thud, an audible slap, and a loud wheeze when expiration or cough forces the object against the lower surface of the vocal chords."[11]

Initial treatment should follow the basic principles of first aid. The time-honoured practice of a sharp blow on the back is frequently effective in dislodging the

foreign body. The patient should be placed on a table or bed so that his head and chest are hanging down.[12] In the case of a small child, grasp him by the feet and hold him up, head down. If this does not dislodge and object, CAUTIOUS digital exploration of the pharynx and epiglottis may reveal a foreign body which can be loosened and manually removed. There is no excuse for hasty or violent probing which may push the object into some less accessible location such as the larynx or trachea. Where the facilities are available the use of a bronchoscope or laryngoscope developed and described by Jackson [13] will provide a surer and safer approach to locating and removing the object. Where severe asphyxia presents and attempts at spontaneous or manual expression fail emergency tracheostomy is indicated. The immediate use of this procedure even where proper facilities are not available may make the difference between life and "aseptic death" to the asphyxiated patient.

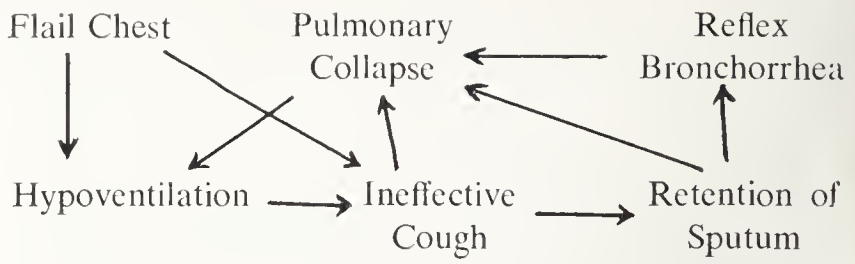
II STOVE-IN-CHEST

"Stove-in-chest", also known as flail chest, is one of the commonest, serious chest injuries. The injury is commonly produced by a crushing force such as that encountered in car accidents where the steering-wheel is pushed back. The force results in a double fracture of one or more ribs, typically in the anterior axillary line or at the chosto-chondral junction and at the chosto-vertebral angle. When several ribs are involved, an abnormally mobile segment of thoracic wall is produced which will tend to sink on inspiration and bulge out on expiration. This may also be seen in the situation where the sternum is fractured along with several ribs on either side. This sign is known as paradoxical respiration. This lesion will, obviously, produce a decrease in respiratory efficiency. The mobile segment will not only reduce expansion of that sector of the lung which it overlies, but the pain of the fracture will discourage movements of the thoracic cage as a whole. Two additional factors will compound the anoxia thus produced:

(i) A unilateral lesion will cause useless shifting of air from one lung to the other thus increasing the effective dead space.

(ii) The decreased respiratory movements will, to some extent, reduce the venous return to the heart which depends in part upon the suction of the inspiratory negative pressure.[2]

The pain and paradox combine to produce other complications which are summarized in a diagram from Grant, Wright and Clifton:[1]



Diagnosis is usually straightforward and can be made on the basis of:

(i) history of crushing injury

(ii) cyanosis, often profound

(iii) obvious pain on respiration

(iv) visible paradoxical respiration

Treatment should be directed along four main lines:

(i) maintenance of adequate airway

(ii) stabilization of the thoracic cage

(iii) artificial respiration

(iv) relief of pain

In cases where severe hypoventilation or obvious accumulations of tracheo-bronchial secretions are present, tracheostomy is indicated. To quote Tabah[5], "Tracheostomy has been shown to be of benefit in this condition in several ways. First of all, it decreases the paradoxical movement of the injured chest segment and in this way decreases pain associated with the movement of the fractured ribs. Second, it provides an easy and ready-made method of aspirating the tracheo-bronchial secretions. Third, there is a decrease in the dead space and also a decrease in the resistance to the inspired and expired air."

The method of stabilization of the flail is the subject of least general agreement. If the lesion is small and the paradox slight the flail can be stabilized by covering with a small pad and strapping with adhesive tape. In the more severe cases, there is a sharp disagreement between the conservative and "immediate surgery" schools. The conservative treatment consists of external adhesive strapping or application of a "many-tailed" bandage.[2] The major danger of this method is that an immediate, and occasionally a permanent, reduction in lung volume may be produced. Surgical fixation is accomplished by traction with towel clips[6] or fixing the ribs posteriorly by suturing the flail segment of rib to the vertebral segment with stainless steel wire.[7] Best method — is assisted respirations, through tracheotomy, in respiratory unit using Bird or other respirator. Positive Pressure breathing — segment floats on lung till stable.

Artificial respiration can be a life-saving procedure and must be applied until the mobile segment can be stabilized. Subsequent ventilation may be necessary if complete stability cannot be achieved. In emergency situations, mouth-to-mouth respiration is effective; in hospital, mechanical assistance, with a demand respirator of the Bennet or Bird type, is usually preferable.[4]

Relief of pain may be at either the systemic or the local level. Narcotic analgesics such as morphine must be avoided as the risk of serious respiratory depression is too great. The liberal use of acetylsalicylic acid and codeine is more effective than is commonly realized.[1] More exacting analgesia may be produced by a paravertebral Novocain block of the thoracic nerve roots.[3]

III SUCKING CHEST WOUNDS

A "sucking" chest wound is one in which a hole, too large to be closed by soft tissues, is produced, allowing free communication between the atmosphere and the pleural cavity. The mechanism of its production is a stabbing perforation of the chest wall. Gunshot and knife wounds are usually sufficiently small that the soft tissues can seal them spontaneously, however, other instruments will produce a hole which is not so easily closed. Car gearshifts are a common example.

The presenting signs of obvious distress and asphyxia are usually very striking. The patient is gasping for breath and is usually deeply cyanotic, unless the associated loss of blood has produced an anemia too severe for cyanosis. If the hole is large enough, a sucking sound is created by air rushing in and out of the wound and bubbles of blood will emerge from it on each expiration. Crepitation may be produced about the wound by air forced into the soft tissues.[6] If mediastinal flutter (to be described later) is severe, a shifting of the trachea with respiration may be observed.

Embarrassment of physiology occurs in three main ways:

(1) Decreased inspiratory expansion of the lungs:

The open communication of the affected side with the atmosphere automatically produces a pneumothorax which is usually severe enough to cause a total collapse of the lung, ie. a loss of 50% of the respiratory capacity.[8]

In addition, the condition of "mediastinal flutter" is usually produced, if there are no previous factors which have caused fixation of the mediastinum and if the hole in the chest wall is as large or larger than the trachea.[9] The ingress of air through the wound on inspiration will cause a shift of the mediastinum to the sound side, preventing normal expansion of the sound lung and further embarrassing the respiratory process. On expiration, the mediastinum returns to its normal position, thus producing "mediastinal flutter."

(2) Interference with circulation:

The shift of the mediastinum on inspiration will diminish venous return by putting pressure on the vena cava and the hilar vessels and, to a certain extent, by kinking the great vessels.[8]

(3) Increase in effective dead space:

As in flail chest, there is a useless shunting of air on expiration from the sound to the injured side when the intra-pulmonary pressure rises above atmospheric pressure. This stale air passes back to the sound lung on inspiration. This dilution of the inspired air further aggravates the anoxia.

Initial treatment of the injury is clearcut: the hole in the chest wall must be plugged. The wound is best closed with a sterile vaseline-gauze pad, however, in desperate cases one should not hesitate to close the wound with any material at hand, regardless of sterility.[6] Improvement of the patient's condition with this simple procedure is usually striking. Later, when proper conditions are available, debridement, control of bleeding and suturing of the wound can be undertaken.

IV TENSION PNEUMOTHORAX

Tension pneumothorax is a condition in which continued escape of air from the lung on inspiration causes a pneumothorax in which the positive pressure in the pleural cavity relentlessly increases. This condition will result from any injury which pierces the lung, leaving the pleura closed and non-adherent.[8] It is commonly seen in knife and gunshot wounds, where soft tissues have closed the portal of entry in the chest wall, and in comminuted rib fractures where a spicule of bone pierced the lung.

The pierced segment of lung will act as a valve, allowing free egress of air to the pleural cavity during inspiration but snapping shut to prevent its return on expiration. The cumulative nature of this process is obvious. The patient literally worsens his condition with every breath he takes. In addition the progressive encroachment of the mediastinum upon the sound lung will steadily reduce the remaining 50% of respiratory function.

Tension pneumothorax should be the first consideration in any patient who presents with no obvious flail or open wound but who is cyanotic and exhibits extreme air hunger and tracheal deviation. Other common but not constant signs are hyperresonance and

absent breath sounds over the affected side, and percussion of mediastinal displacement.

A therapeutic diagnosis can be made by inserting a large needle into the pleural cavity. The escape of air under pressure is diagnostic of a tension pneumothorax and positive pressures of 15-20 cms. of water, are often found.[8] In severe cases, any form of pressure release, even a knife stab, is preferable to delay. This can be accomplished in several ways. One method is with an indwelling needle which is fixed in position with a cork strapped to the chest wall and which is covered by a rubber "flutter valve."[6] Another is by the insertion of a large catheter (eg. a 24 Jaques) through the second intercostal space. The catheter is connected to an underwater-seal-drainage bottle and negative suction may be applied.[1] The second space is preferable for both needle and catheter aspiration as the escaping air will tend to occupy the uppermost part of the pleural cavity.[7] If severe air leak persists, there is probably damage to the trachea or to a major bronchus. Thoracotomy and operative repair of the leak will probably be necessary.[1]

I have tried to emphasize in all the above conditions that prompt instigation of specific first-aid measures is of infinitely more value than the marshalling of spectacular but delayed surgical procedures. The prognosis for a complete recovery is good in all, if the physician will act swiftly to avoid the permanent damage of anoxia.

Summary

Four conditions representative of emergency chest injuries are discussed and immediate first-aid management is described and advocated.

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(cont. from page 90)

(3) It can be used for nodal, atrial, and ventricular tachycardias as well as atrial and ventricular fibrillation.

(4) In Lown's experience there has been no case of ventricular fibrillation or cardiac arrest. The only complication was a single episode of systemic embolus 24 hours after restoration of sinus rhythm. Dr. J. K. Wilson (St. Michael's H.) points out that to date 10% of patients with ventricular tachycardia and atrial flutter have been successfully treated. Over 80% of patients with atrial fibrilation can be successfully restored to normal rythm. In this latter group, however, there is a disappointing recurrence rate in spite of maintenance therapy with quinidine or Pronestyl.

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Emergency Treatment of Burns

H. G. Walker 6T5

INTRODUCTION

It behooves all members of the medical profession to be familiar with the emergency treatment of burns. The home, industry and rapid transit of to-day contribute yearly to an increasing number of burn victims.

Furthermore, in the event of a third world war, this would more than likely be thermo-nuclear in type and consequently the number of burn casualties great. Medical establishments of the Armed Forces and civilian hospitals are putting up an intense effort to prepare themselves for such an emergency by the proper training of personnel and the stock piling of various equipment.

It is not the purpose of this short article to discuss the criteria and methods of treatment in the event that a large number of burn casualties present themselves at one time. I shall merely concern myself with the isolated case and outline the best method of dealing with it therapeutically.

General Considerations

It is imperative that whoever attends a burned patient or even comes near him should observe sterile precautions, and be dressed to this effect with cap, mask, gown, and gloves when handling the burned area. This obviously is to prevent further contamination than the patient may have already suffered during transport and handling from the scene of the accident to the treatment center.

The objectives in burn management are:

1. Prevention and treatment of shock.
2. Prevention of infection.
3. Relief of pain and apprehension.
4. Healing of the wounds.
5. Preservation of function.

Clinical Assessment of the Patient

a. History

Determine when the victim was burned and under what circumstances.

What was the patient's health before the accident?

Were any narcotics or other medications given? If so, what and how much?

b. Physical

(1) Inspect the mouth, nose and pharynx. Remove any foreign material or objects. Determine whether this area has been burned. Provide an adequate airway.

(2) Carefully remove clothing or first aid dressings; cut away if necessary, in order to prevent unnecessary moving of the patient. Treat shock if already present. Be wary of entrance into shock even after admission into hospital.

(3) Determine the extent and the degree of the burns and whether or not there are any other injuries which need immediate attention.

The extent of the burns may be obtained by applying Wallace's "rules of nine", thereby expressing the percentage of total body surface area burned: B.S.B.

Head and neck	9%
Back	18%
Front	18%
Arms, each	9%
Legs, each	18%
Genitalia & perineum	1%

This does not apply for children under the age of seven years. Clarkson ([1]) states that a "rules of six" is more applicable at age 2-3 years, in that the lower limbs are designated 12% each, the upper limbs 6% each and the trunk, head and neck obtain an appropriate increase in the percentage of body surface area as compared with the adult.

The depth of the burns is not easily assessed in the first few hours after the injury. Nevertheless, it is important to estimate the depth of the burns, since obviously deep burns are more serious than superficial ones and treatment must be planned accordingly.

First degree burns are red in appearance, blanch under local pressure, produce a minimal amount of oedema, if at all, and display hyperesthesia.

Second degree burns usually display an intact or broken blister, oedema and hypesthesia.

Third degree burns are usually dry, whitish or charred, and demonstrate anesthesia.

Clinical Procedures and Treatment

(1) Venipuncture. Use a large gauge needle. Collect blood for cross and type, hemoglobin, and hematocrit and serum electrolyte determinations.

Leave the needle in place and start rapid infusion of lactated Ringer's solution. Laboratory procedures should be repeated every four hours as necessary.

(2) A vein cut-down, followed by the introduction of a cannula is advisable in children, patients in shock and those who are to be transported.

(3) Catheterize the patient if comatose, unable to void, or his condition grave. Make careful observations as to the nature and the amount of urinary output. Evidence of hemolysis may accompany acute renal failure. ([2])

(4) For the relief of pain and apprehension morphine or other narcotics may be given, but must be administered IV, since the status of the vascular complex in muscle or skin is doubtful in these patients and overdosage is a danger.

Dosage: Morphine Sulphate Solution IV, 8-10 mg for adults, Morphine Sulphate Solution IV, 0.1 mg per pound body weight in children.

(5) Colloid and fluid therapy. Enter all observed and estimated qualities and quantities of fluid loss and fluids administered on a fluid balance chart. If the haematocrit is above 50, blood may be withheld until other colloids, such as Dextran have been given. Patients with severe burns should have two-thirds of the quantity of colloids administered in whole blood.

Fluid requirements may be estimated roughly by applying Evan's formula ([3]) which calls for: 1 c.c. each of colloid and electrolyte solution per 1% B.S.B. per kg body weight, with an upper limit of 3500 ml of each on the first day and half this amount on the second day. To this should be added 2000-3000 ml per day of oral fluids to meet normal metabolic requirements.

A strong word of caution here is appropriate. One must not adhere too dogmatically to an emperical formula such as this. It is to be used as a guide. The patient's blood pressure, respirations, pulse rate and level of consciousness, by and large, remain the most useful guides to the patient's condition. Though clinical judgement, careful observation and strict attention to ever changing therapeutic requirements often defy objective analysis, yet they are the factors on which success or failure largely depend in the management of severely burned patients.

Special Considerations

Infection:

Most authors agree from the results of independent studies, that the administration of antibiotics to all severely burned patients reduces the mortality. Sako ([4]), in a recent review of the emergency treatment of burns includes prophylactic antibiotic coverage of either penicillin and streptomycin or a broad spectrum antibiotic, given for four or five days to all critically burned patients.

Patients with severe burns who have previously received active immunization against tetanus should be given 0.5 cc of toxoid. Those not previously immunized are given 5000 units of anti tetanus serum, observing the usual precautions for hypersensitivity reactions. Patients with less severe burns, not previously immunized, should be started on active immunization with tetanus toxoid.

Shock:

Patients in shock can be treated best by stepping up their rate of fluid infusion. If the patient is seen three to four hours after the burn, without having received treatment, the amount of fluid by calculation scheduled for administration for the first eight hours may be given in from four to five hours, but with caution.

The urine output for adults should, if possible, be maintained at 35-50 cc per hour, and for children at 20-25 cc per hour. ([5])

Respiratory Tract Burns:

In patients who have received burns to the respiratory apparatus a tracheostomy is indicated. This measure provides the patient with a clear airway, reduces the physiological dead space by some 170 ml, makes humidification of the inspired air easy, facilitates the removal of secretation and ensures that immediate mechanical assistance can be rendered to the patient's breathing. ([6])

Auscultation of the chest must be done frequently if possible in order to detect the presence of pulmonary oedema which requires immediate alteration in the patient's fluid therapy.

Definitive Treatment of Burned Area

Many authors agree that the exposure method is the method of choice in the management of burn cases. Dupont et al ([7]) do not carry out a debridement of the burned areas in their patients for about two to three

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Status Asthmaticus

P. W. Munt 6T4

Status asthmaticus is a "condition in which bronchial asthmatic attacks become continuous with little or no remission by day or by night and do not appear to respond to any form of therapy."[1] This condition is to be differentiated from the acute paroxysms of asthma which are readily amenable to the more routine methods of medical therapy.

In status asthmaticus the patient has usually been treated with epinephrine subcutaneously, nebulizers containing either epinephrine or isoproterenol, sedation in the form of pentobarbital sodium, or phenobarbital and usually aminophylline intravenously, or rectally, but to no avail. The patient progresses to extreme anxiety, orthopnea and dyspnea, with cyanosis and impending asphyxiation. The following is a typical patient:

Mrs. R.A.: A forty year old woman was admitted to hospital in December 1960 with severe dyspnea and cyanosis. She was a known asthmatic for twenty-five years with minor attacks five to six times per year and six major attacks which required hospitalization.

Physical Examination: Respiratory rate of thirty per minute. Increased use of accessory muscles of breathing. Increased resonance on percussion of chest and palpable rhonchi. Auscultation revealed wheezing throughout both lung fields with sibilant rhonchi bilaterally and prolonged expiratory phase of respiration. No rales were heard on inspiration.

Lab: Hemoglobin — 14.5 G. Hematocrit — 48%. White Blood Count — 8,000. Blood Urea Nitrogen — 15mg.%. Differential — increased number of eosinophils (25%). Sodium — 128 m.eq. 1L. Chlorides — 98 m.eq. 1L. Potassium — 4.0 m.eq. 1L. Serum Bicarbonate — 21 m.eq. 1L.
Urine — ph 5.5 2 + sugar 2 + acetone

X-Ray: Both lung fields clear with slight decrease in parenchymal density; heart shadow normal; slightly increased bronchovascular markings.

Regimen: For three days this patient had been treated at home with epinephrine subcutaneously, sedation consisting of phenobarb, short-acting tranquilizers, and chloral hydrate, plus aminophylline, steroids and penicillin. Despite this therapy the patient's dyspnea increased; she became more cyanosed and very fatigued with aching of her chest and abdominal muscles. Thus she was refractory to her present medical regimen which had to be altered and she was now in true status asthmaticus.

Pathology: Death is rare from uncomplicated asthma; the usual case of death is *asphyxia with terminal bronchopneumonia*. Gross examination of the lungs shows they are voluminous filling the entire thoracic cavity and there are often white mucous plugs filling the smaller bronchi.

On microscopic examination there is thickening of the walls of the bronchi and bronchioles, with thick tenacious mucus in the lumina. Usually the trachea and main bronchi are unaffected although there may be some reddening of the mucosa. Thickening of the bronchioles and bronchi is due to edema and cellular infiltration. The bronchial muscle is contracted and there is a usually hypertrophy. The epithelium shows increased cellularity and the submucosal glands are hypertrophied and filled with mucin. There is also hyaline thickening of the basement membrane.

The lung parenchyma may show emphysematous changes with rupture of the alveolar walls.

Signs and Symptoms: These people present with severe dyspnea and orthopnea with or without cyanosis. There is an over-distension of the chest, often an audible wheeze and they may complain of aching of the chest and abdominal muscles. There is hyperesonance on percussion, decreased diaphragmatic excursion and loss of cardiac dullness. Auscultation reveals a prolonged expiratory phase productive of sonorous and sibilant rhonchi.

These patients are extremely restless and anxious partly due to fear, and partly due to carbon dioxide narcosis. There is usually a history of previous asthmatic attacks or allergy in the patient or family.

Particular attention should be paid to the sputum if present; normally it is white and mucoid although it may be blood streaked in status asthmaticus and is yellow if an infective process is present. Infection may also manifest itself by fever, leucocytosis and raised sedimentation rate, but frequently the white blood count, and sedimentation rate are normal, and fever is absent.

Presence of cyanosis must be noted and cautious oxygen therapy commenced. Evidence of right heart failure may be present if the patient is old or has chronic asthma with emphysematous changes.

Signs of dehydration and shock must be corrected immediately.

Differential Diagnosis: Usually there is no problem in making the diagnosis. However, cardiac "asthma" must be considered, in which there are inspiratory moist rales, bloody sputum and cardiomegaly.

Poisoning with cholinergic drugs or insecticides cause bronchospasm with a similar picture which is relieved by atropine. Carcinoid tumours with increased serotonin levels, cerebral tumours and foreign body aspiration can usually be ruled out.

Laboratory Findings:

Blood: Hemoglobin —should be normal or slightly evelvated due to hemoconcentration if dehydration has occurred.

White blood count —Normal unless infection present.

Smear —Normal, increased eosinophils (30%) and atypical lymphocytes in the differential.

Blood Urea Nitrogen —should be normal.

Electrolytes pCO_2, may be elevated and serum bicarbonate decreased.

Urine: —Volume decreased if dehydration is present.

pH —Decreased; ketones may be present if the patient hasn't eaten and is breaking down fat stores. Sugar may be present if the patient has received much adrenalin, adrenal steroids or intravenous glucose.

Sputum:Smear —shows eosinophilia and organisms if infection is present as is usual in status asthmaticus. Culture and sensitivity may reveal normal flora.

Unstained Smear —Shows the so called Curschmann spirals which are twisted spirals of mucous from bronchioles; Charcott-Leyden crystals may also be seen which are probably derived from the nuclei of cells.

E.K.G. —May be normal or in chronic asthmatics there may be right ventricular hypertrophy .

X-Ray: Chest films may be normal or may show emphysematous in both lung fields, flattening of the diaphragmatic domes, increased AP diameters of the chest.

Heart shadows are normal unless there has been longstanding pulmonary hypertension due to emphysematous lung changes with right ventricular hypertrophy.

Treatment:

All patients must be hospitalized and nursed in the orthopneic position since the abdominal musculature relaxes permitting further descent of the diaphragm and increased air entry to the chest. Much reassurance is necessary both to the patient and to his family.

1) *Sedation*: This plays a very important role in these patients. They are extremely anxious people, many of whom are panic-stricken with the fear of suffocation. Because of their original shortness of breath they develop anxiety which in turn causes further hyperpnea forming a vicious circle which must be broken by the use of a good sedative. They are also very fatigued from their over breathing, and have an aching of their chest and abdominal muscles. Also, once they are sedated it allows the physician sufficient time to plan an adequate course of therapy without undue haste. MORPHINE must never be used! It can aggravate and depresses the cough reflex and respiratory centre with resultant accumulation of bronchial secretions and further anoxia. Although demerol has been advocated by some as well as phenobarb, these drugs are unsuitable in status asthmaticus.

The most suitable drug is ether: 2 oz. mixed with olive oil, 2 oz., and given rectally. This usually sedates the patient for several hours and may be repeated when necessary.

Tranquilizers may aggravate impending shock and barbiturates, such as pentobarbitone, may produce restlessness in the patient.

2) *Intravenous Therapy*: This is important in the management of a patient extremely ill and unable to take fluid per os. Intravenous fluids are necessary to maintain hydration and nutrition, to aid in liquifying viscid secretions, and to act as a vehicle for drugs.

Regimen: (A) Commence with one litre of 5% glucose and normal saline. Usually one bottle (of saline) per

24 hours is sufficient to correct eletrolyte and acid-base imbalance. This litre can be given in the first few hours.

(B) Within the next sixteen hours two or more litres of 5% glucose in distilled water may be required.

The only dangers with intravenous therapy are cardiac overload and increased hypernatremia if there is evidence of heart failure. Molar lactate has been advocated by some to decrease the acidosis; subsequently the bronchi and bronchioles may become more sensitive to the effects of epinephrine.[2]

3) *Steroids*:

Hydrocortisone Sodium Succinate—100mg, intravenously STAT and then add 100 mg to 1000 c.c of the first bottle of the intravenous solution. ACTH is not used since the adrenals have already been greatly stimulated and because the patient may have been on steroids prior to admission with the result that there will be little response to stimulation.

Oral Steroids are commenced once the patient can tolerate them. Commence with prednisone (or a corresponding drug):

15 mg. q6h the first day
10 mg. q6h the second day
5 mg. q6h thereafter for a week providing the patient is responding well to this therapy.

4) *Oxygen*: Judicious use of oxygen can be of great benefit if the patient is cyanosed. Intranasal catheter is probably the best method. 100% oxygen is given at the rate of five litres per minute. (20% Helium and 80% Oxygen may be substituted). Use of a mask or tent tends to create further anxiety in already fearful patients. Using a nasal catheter also allows one to use ether and oil at the same time, whereas there is danger of explosion when an oxygen tent and ether are used.

Other precautions must be taken. These include oxygen poisoning and cessation of respiration. Because of CO_2 narcosis the only stimulus to respiration is hypoxia acting on the carotid and aortic bodies. When oxygen is administered too vigorously there is no stimulus to breath since the hypoxia is rapidly corrected while the carbon dioxide levels remain at narcotic amounts in the blood. In this case respiratory stimulants are necessary.

5) *Antibiotics*: These are used if there is any evidence of pneumonitis as suspected by pyrexia, purulent sputum, leucocytosis and an elevated sedimentation rate. In fact most cases of adult status asthmaticus are due to infective asthma, occassionally attributable to pneumococcus, Klebsiella pneumoniae or staphylococcus, but usually initiated by a virus infection. It is best to start with Tetracycline 500 mg. t.i.d orally. If the patient is unable to swallow or is in impending shock in which case intestinal motility and absorption will be greatly decreased, Tetracycline must be given parenterally - 500 mg. b.i.d.

Penicillin is a potentially dangerous drug to give parenterally since many of these allergic individuals will be sensitive to it.

6) *Expectorants*: Drugs such as potassium iodide and ammonium chloride are advocated by some to liquify bronchial mucus. These are of little value in status asthmaticus and are of questionable value in the recovery phase.

7) *Radical Treatment*: If all the above measures fail, which should never occur, unless the patient has been brought to hospital too late, the patient must be sent to the respiratory unit of a large hospital or one should get the aid of an anesthetist.

One author [3] suggests a method of alternate positive pressure breathing and endotracheal tube suction:

(a) The patient's lungs are first ventilated manually by a bag and mask using intermittent positive pressure breathing.

(b) After several minutes gastric decompression is achieved with a lavage tuge.

(c) An endotracheal tube is passed.

(d) Alternate tracheobronchial suction for 30 seconds followed by a three minute ventilation period. This cycle is continued for one and a half to four hours until all obstructive material is removed and good spontaneous respirations are achieved. In some cases tracheostomy may be necessary.

8) *Subsequent Therapy*: As the patient is coming out of status asthmaticus he may become increasingly responsive to epinephrine and aminophylline. Aminophylline can be given in the dosage of 0.5 G in the intravenous drip and epinephrine can be given 0.2 c.c subcutaneously as required.

Bronchodilators such as epinephrine inhalation (1:100) or isoproterenol used as nebulizers taking one to three inhalations every 10-30 minutes, may now be of some value.

However, care must be taken in the use of epinephrine injections or vapour nebulizers in cases of

hypertensive disease, and angina pectoris. Moreover, in excess amounts nebulizers can cause severe irritation of the brochial mucosa.

The use of oral steroids has been outlined above in the initial treatment. However, if the patient is a severe chronic asthmatic, then the benefits of maintenance prednisone may outweigh the dangers.

Prophylaxis:

(1) *Eliminate allergens*: especially those known to aggravate the person's asthma e.g. pets, feathers, pollen, cigarette, and cigar smoke, flour dust, etc. Some patients are sensitive to bacteria or bacterial products; focal areas of infection such as chronic sinusitis must be drained adequately at first by medical and then, if necessary surgical means.

If chronic infective asthma is the cause, tetracyline may be given continuously during the winter months to decrease the frequency of attack.

(2) *Drugs*: These are given to the patient to abort attacks. There are many drugs but most contain ephedrine (25 - 50 mg.), phenobarb (15 - 60 mg.), and aminophylline (0.25 - 0.05 mg.) or a combination of these.

Nebulizers containing epinephrine or isoproterenol may be used observing the above precautions. Occasionally an alert and intelligent patient, can be taught to give himself epinephrine subcutaneously.

(3) *General*: In most patients psychological factors play a part in the disease, and psychiatric consultation may occasionally be necessary. Extremes of cold and humidity must be advoided. These people must exercise only to the point of tolerance. In some, breathing exercises stressing prolonged and complete expiration rather than deep inspiration may be of value especially in preventing chest deformities which are more apt to occur in children and adolescents. Specific desensitization is less effective with asthma than hay fever and often improvement does not persist when the treatment is discontinued. The patient must be on a well-balanced diet, and should avoid obesity and fatigue.

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weeks and the patients are kept under the best possible sterile conditions in their beds without dressings. By this time second degree burns will have healed and the patients can be prepared for grafting of the third degree burn areas.

Sako states that after shock has been controlled the patient is moved to the operating room where the burned areas are gently soaked with sterile saline solutions and under analgesia the burned area debrided. For very soiled areas the soaks are kept up somewhat longer. The patient again is left without dressings. This debridement procedure may not be possible in electrical burns where the extent of damage is not limited to the visible burn area and more extensive excision may be needed due to permanent vasospasm and consequent necrosis of distal areas.

If a dressing is needed, as it may be in circumferential burns, Furacin or some other antibiotic ointment may be employed. The immediate goal at all times is to maintain a clean wound and to prepare areas for staged grafting as necessary. The ultimate goal is to lessen morbidity and mortality; to rehabilitate the severely burned to satisfying useful activity.

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Acute Appendicitis

A. C. Gold 6T4

Introduction

Acute appendicitis is one of the commonest clinical diagnosises for which the abdomen is opened; or as an American text states, "acute appendicitis is the commonest cause of right lower abdominal pain in all ages."[1] Needless to say, no matter if it is in a large city hospital or in a county clinic, this cause of lower abdominal pain must be put in the fore of all differential diagnoses.

It is important to realize that this pathological state is a clinical diagnosis in every instance, and the confirmation or reinforcement with laboratory tests is purely as such, and should not significantly alter the doctor's approach to management in the acute stage.

This article will deal mainly with the diagnosis, treatment, differentiation of perforated and non-perforated appendicitis, and important clinical entities to be considered and excluded before diagnosis and management are undertaken.

Diagnosis of Non-Perforated Appendicitis

Classically, there is a history of crampy periumbilical pain shifting to the right lower quadrant in twelve to twenty-four hours associated with nausea, vomiting, and fever. On physical examination there is hyperasthesia, and maximum tenderness over the right lower quadrant with rebound and rectal tenderness in the same area.

Due to variation in the appendical position, there must be adequate allowance for different points of maximum pain, tenderness and hyperasthesia. A retrocaecal appendix may give a diffuse right lower quadrant pain, but rectal tenderness is equisite. In pregnancy, the appendix may be displaced somewhat superiorly and lateral to its normal position with corresponding symptoms and signs in this area. And a low lying pelvic appendix may irritate the bladder with resulting red cells in the urine. Although acute appendicitis is uncommon in children under 2 years of age, yet when it does occur, the most consistent sign is abdominal distension. Anorexia, nausea, vomit, fever, and leukocytosis are highly variable and should not substantiate a diagnosis in themselves.

Perforated Acute Appendicitis

The sequelae to perforation are local or diffuse with either the pus and fecal material walled off in an abcess or free in the peritoneal space. The signs and symptoms are always localized to the right lower quadrant at first but after a variable period of time they may or may not become diffuse with signs of generalized peritonitis. A mass in the right lower quadrant is almost diagnostic of a perforated appendix in a younger person but in the middle-aged or elderly patient, one must entertain the possibility of carcinoma of the caecum.

Pain is always localized at first and may temporarily abate because of destruction to the nerve plexus in the appendiceal wall from distension or rupture. Tenderness always increases with perforation and the abdominal muscle becomes rigid but not to the board-like rigidity that accompanies perforated peptic ulcer. Bowel sounds are decreased to absent, and the laboratory results are non-specific, there being a high white blood count and sedimentation rate. Rarely is there free air extruded into the peritoneal cavity; therefore, the flat plate of the abdomen is normal.

The incidence of this complication is higher in infants due to late diagnosis and surgical intervention; and in the elderly where there is decreased sensory perception of pain and poor muscular tone obscuring local or generalized rigidity.

Differential Diagnosis

Although it is possible to classify on a symptomatic or physiological basis, an anatomical classification is probably most workable and may be done so under the following scheme.

(A) *Right Adenexae*

1) Twisted ovarian cyst or tumor
2) Ruptured graffian follicle
3) Ectopic pregnancy
4) Torsion of the right fallopian tube
5) Right unilateral P.I.D.
6) Acute salpingitis

(B) *Caecum*

1) Diverticulitis
2) Carcinoma
3) Non specific ulceration

4) Torsion of appendices epiploicae

5) Distension of caecum secondary to large bowel obstruction

(C) *Ileum*

1) Crohns disease

(D) *Retroperitoneal*

1) Right ureteral stone

2) Right pyoureter with extension to the peritoneum

3) Acute urinary retention in children

(E) *Others*

1) Those causes in the abdomen:

(a) Perforated peptic ulcer

(b) Acute cholycystitis

(c) Mesenteric adenitis

(d) Acute pancreatitis

(e) Hepatic and sub-hepatic abcess

(f) Extra-peritoneal hematoma

(g) Mesenteric thrombosis

(h) Intra-abdominal herniation

(i) Ruptured abdominal wall

2) Local or generalized conditions referred to abdomen:

(a) Right-sided pleuritis

(b) Cord and vertebral diseases

(c) Herpes Zoster

(d) Tabes dorsalis

(e) Coronary thrombosis

(f) Rheumatic fever

(g) Porphyria

(h) Sickle cell anemia

(i) Periartertis nodosa

Treatment

(A) *Non-Perforative*

The treatment of acute appendicitis is appendectomy carried out as soon as the diagnosis is made, providing the patient is in good general condition.

Following an appropriate anaesthetic, a gridiron incision is made in the right lower quadrant, the appendix is identified, freed from surrounding tissues, if necessary, and the base is clamped and the vessels ligated. The appendix is then removed and the stump is closed in one of several ways, each to the surgeon's preference. If there is the question of slight rupture and spillage of pus, then a small soft rubber Penrose drain may be left in and carried to the outside through a lateral stab wound. Or the drain may be placed subcutaneously, if there is the possibility of wound infection and brought out through the lower end of the original skin incision.

Without complications, the patient is ambulant the day of the operation, and is usually placed on a soft diet for several days.

(B) *Perforative Acute Appendicitis*

It is in this facet or complication of the disease that opinions differ highly as to management. Some surgeons argue in favour of conservative therapy for the reasons that the abcess wall will not be disturbed and that there is the possibility of an immune antibody formation that will counter the infection. Some also feel that there is no sense putting the patient under increasing strain with prolonged time under anaesthesia. Others vehemently disagree and say bluntly that it is foolish to allow the source of infection to remain in the abdomen. If the patient's clinical course was worse before seen, then conservative therapy preceding surgery is usually carried out; or if there are symptoms and signs of multiple abcesses having developed, treatment is basically one of vigorous antibiotic therapy.

Mosley favours this form of management in most cases of perforative acute appendicitis and he follows the so-called Oschner-Sheron regime:

(a) complete bed rest.

(b) notthing by mouth for 3 to 4 days.

(c) I.V. 5% glucose in saline solution.

(d) antibiotics, penicilin and streptomycin.

(e) blood or plasma transfusions, if necessary.

The pulse rate is charted every two hours and with any significant increase in the rate, or at the first signs of vomiting, surgical intervention is deemed necessary. Without further complications, the patient is allowed to go home but returns in several months to have his appendix out.

(C) *Other Abdominal Conditions*

If appendicitis occurs in pregnancy the inflammatory reaction, if allowed to continue, may irritate

the uterus and initiate labour or an abortion. If an appendiceal abcess forms, the uterine contrations may lead to rupture of the abcess. There is little justification for conservative therapy with appendicitis in pregnancy, for the gamble of initiating labour with inadequate antibiotic therapy far outweighs the difficulty of surgical intervention. The localized signs and symptoms are more lateral and superior due to the displacement of the appendix, and the surgical approach is correspondingly altered.

The removal of an appendix not diseased or symptomatic in the course of abdominal surgery is purely elective. Most surgeons will remove it with the idea of prophylaxis in mind, yet a few hesitate on the grounds of unnecessary procedure and increased risk.

Complications

The appendix differs little from the remainder of the bowel as to its complications which are obstruction, perforation with abcess formation and/or generalized peritonitis. Perforated appendicitis is the most common cause of bacterial peritonitis, and it may be further complicated by hypovolemic shock. The actual incidence of localized peritonitis, or abcess formation is difficult to assess because many surgeons employ conservative methods of treatment.

Pylephlebitis is a most serious complication, but fortunately, it is rare. There is usually a septic thrombophlebitis with emboli from the appendix. The septic clots may shower emboli into the liver causing multiple abcesses. The onset of recurring chills with mild jaundice, and tenderness over the liver points strongly to this diagnosis. After a blood culture has been taken, massive antibiotic therapy is instituted and if unsuccessful, surgical exploration and drainage is the next step in management.

In the case of paralytic ileus which accompanies a perforated appendix with generalized peritonitis, treatment is conservitive with massive antibiotics, intravenous fluids, and a Levine tube for drainage.

Other complications are: wound infections, fecal fistula, hernia, pulmonary embolus, and those of anaesthesia.

Prognosis and Mortality Rate

From 1927-41, the University of Chicago Clinics found that the death rate from appendicitis and peritonitis was 17/2550 autopsies, while there were only 2/2450 from 1941-52. While the mortality rate has strikingly decreased, the morbidity has remained the same and even increased somewhat. One can account for this by realizing that the introduction of antibiotics, blood and plasma transfusions coupled with earlier diagnosis and treatment surgically will reduce the death rate, yet those saved are now the victims of septic complications.

The physician must train himself to diagnose the causes of localized peritoneal pain for in the case of acute appendicitis, this is an emergency because there is the possibility of perforation; but once perforation has occurred the emergency situation has passed, and treatment of the complications begin.

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Medical Shock

P. G. Strachan 6T4

Introduction

Shock may be defined as a failure of the peripheral circulation characterized by a reduction in the effective circulating blood volume and in the blood pressure. Clinically, the patient has an ashen-gray, pale appearance and beads of perspiration hang from his forehead. He is very apprehensive and restless, and will complain often of a dry mouth and thirst. With any physical exercise, he rapidly weakens, as the exchange of oxygen and nutrients has become impaired by the reduction of effective blood volume. His pulse is rapid and thready; his breathing is rapid and shallow. He must be recognized and treated with speed and efficiency.

Medical shock is defined in this article as any type of shock which is not produced by blood loss. There are two major types of medical shock a) primary and b) secondary. Primary shock is synonymous with simple syncope or fainting, and is a temporary circulatory inadequacy resulting from vasovagal reflexes. Neurogenic or psychic factors appear to precipitate the attack. For example, some people faint at the sight of blood or the anticipation of an injection. Such syncope is temporary and is of little clinical importance. Secondary shock is of great importance to the physician. There is always some clinical or metabolic derangement which requires immediate and adequate treatment.

Physiologically, an inadequate circulation (secondary shock) may be produced in many ways. Stead suggests the following physiological classification:

1). inability of the heart to pump the needed blood due to weakness of the heart itself. (examples— myocardial infarct, paroxysmal auricular tachycardia, atrial flutter, rapid atrial fibrillation)

2). failure of proper filling and ejection of blood because of pericardial tamponade.

3). reduced venous return. (examples — blood loss, plasma loss in burns, peritonitis, anaphylaxis and dehydration, especially with salt loss)

4). obstruction of main arterial pathways, as by pulmonary embolus.

5). widespread failure of cell metabolism

6). loss of vasoconstrictor tone. (anaphylaxis, sodium nitrite ingestion, septicaemia)

One or several of these physiological changes in the circulation may be acting in any given case.

To discuss all types of medical shock in this article would be an impossible task. As a result, I have chosen three representative types, and have attempted to present the special problems associated with each. Septic shock is of special interest because of a rising incidence. Pulmonary embolus and cardiogenic shock are of importance because of the frequency with which they are met clinically.

SEPTIC SHOCK

Etiology and Incidence:

Because of an aging population and more extensive surgery, there has been a rising incidence of septic shock. Lansing defines septic shock as "a state of hypotension caused by bacteremia and accompanied by oliguria, signifying decreased peripheral blood flow." Two-thirds are caused by Gram-negative organisms such as E. Coli, Aerobacter aerogenes, Proteus and possibly Pseudomonas, while one-third are caused by Gram-positive organisms such as staphylococcus or streptococcus.

A series of 150 patients at the University of Minnesota Medical Center showed that 90% of cases developed in males. In this same group, there was a mortality of 70%. This condition was a rarity in the paediatric age group, except in newborns and patients with meningococcemia. The predominance of the male indicated the importance of urinary infections resulting from obstruction, as the underlying cause. The high mortality demonstrates why early and adequate medical therapy is required in all cases. However, it must also be pointed out that only about 20% of all patients that develop a bacteremia will actually go into a state of shock, and it is in this group that the high mortality exists.

Clinical Aspects:

The most important signal of impending septic shock is a shaking chill. In addition, there is generalized malaise, aches and pains, fever (unlike many shock states) and leukocytosis. A good urinary output will exist in the warm, dry hypotensive while a low output will be present in the hypotensive with a cold, clammy skin. Both clinical states may occur, the former sometimes progressing to the latter less favourable condition.

Treatment and Rationale:

The pathogenesis of septic shock is unknown. One theory suggests that histamine release is the local

effect of endotoxin, while its general effect is adrenaline and nor-adrenaline release from the adrenal medulla. Gradual exhaustion of the medulla and increasing concentration of histamine results in vasodilatation, a decline in peripheral resistance and sludging of blood. Another theory suggests that a state of hypersensitivity, either toxic or allergic, is the main pathogenesis.

Both theories support the use of adrenocorticosteroids, at least on a theoretical basis, as this hormone suppresses the action of histamine and combats hypersensitivity reactions. The postulation that improvement following cortisone administration is due to a failing adrenal cortex has no evidence to support it.

In a patient developing septic shock, the most important first step is to start an I.V. to act as a means of administering drugs. A blood culture is obtained as well as a culture from elsewhere if there is a discharge or some other sign indicating a site of infection. Intravenous antibiotics are started immediately. The choice of antibiotic is based on the clinician's suspicion as to the most likely organism. For Gram-negative septicemia, the following drugs may be used;

a) bacteriostatic agents, such as tetracycline or chloramphenicol

b) bacteriocidal agents such as erythromycin or penicillin. It must be remembered that more than one organism may be involved.

In the presence of definite hypotension or oliguria, vasopressors such as noradrenaline or metaraminol are given, the former being more effective but requiring very close supervision. Rogitine in the bottle with noradrenaline will minimize the chances of skin necrosis without interfering with the vasopressor effect of the drug. Blood pressure and urinary output must be closely watched.

Three hundred to 500 mg. of hydrocortisone every 8 hours for 3-5 days may be given in the severely ill patient for reasons previously mentioned. Most authors feel that this therapy can do no harm and may improve the patient.

Intravenous solution should replace only measured losses and meet the metabolic requirements of the patient in the presence of oliguria, as fluid therapy may result in pulmonary edema if the patient is overloaded. Metabolic acidosis should be corrected with sodium bicarbonate solution.

The most important aspect of therapy is speed. The sooner the patient is treated, the greater are his chances of survival. Septic shock, with its high mortality, provides the grounds for a great deal of further investigation in the fields of therapeutics and pathophysiology.

PULMONARY EMBOLUS

Etiology and Incidence:

Most pulmonary emboli are detached thrombi from the venous system or from the chambers of the right heart. On occasion, the embolus may consist of fat, amniotic fluid, air, bone marrow, tumor or foreign body. This discussion will be limited to blood clot emboli.

The source of 60-80% of pulmonary emboli is the peripheral veins in the lower extremities. For unexplainable reasons, emboli originating in the arm veins are rare. Other relatively common sources are pelvic veins (postpartum, following surgery or following septic abortion), the prostatic plexus (following surgery) and right heart chambers. (atrial fibrillation, myocardial infarction, endocarditis etc.)

The actual incidence of pulmonary embolus is impossible to assess, as many are not sufficiently large to produce infarction or symptoms.

Clinical Aspects:

The following signs and symptoms may occur:

1). sudden dyspnea
2). substernal pain
3). right-sided heart failure
4). tachycardia
5). restlessness
6). anxiety
7). syncope
8). hypotension

In addition, the physician may notice pulsation in the second left interspace, an accentuated "second pulmonary sound," pleuropericardial friction rub, and murmurs in the second left interspace. Investigation should include the following—

1). history and physical examination with a careful enquiry directed towards locating the source of the embolus.

2). serial electrocardiograms, which will reveal transient changes in most instances.

3). chest X-ray, which may show evidence of pulmonary infarction.

4). laboratory values—leukocytosis, increased ESR, increased lactic dehydrogenase with a normal SGOT and an elevated serum bilirubin.

The severity varies from sudden death, which will result from rapid occlusion of both pulmonary arteries (a saddle embolus) to a mild, symptomless emboliza-

tion involving minute pulmonary vessels. A state of shock may occur, with death ensuing in a matter of hours or gradual recovery over a number of weeks. The diagnosis is made in only 20-50% of cases, as confirmed by autopsy. The differential diagnosis includes:

1). acute myocardial infarct
2). pulmonary edema
3). atelectasis
4). pericarditis
5). spontaneous pneumothorax
6). ball valve thrombus (left atrium)
7). dissecting aneurysm
8). pulmonary artery thrombosis

Treatment:

The physician's hands are tied in the acute stage of pulmonary embolus. A few reported cases of successful embolectomy or fibrinolysis exist, but this treatment is presently not satisfactory. The aim of therapy is to prevent recurrences and to provide symptomatic relief.

Analgesia for chest pain is important. Demerol is preferable to morphine as it causes less respiratory depression. Oxygen is given as indicated for dyspnea and cyanosis. Thoracentesis may be necessary to allay dyspnea and may be helpful in making a diagnosis. Laxatives will reduce straining at stool, which could precipitate the detachment of further emboli. If severe, vasopressors intravenously (noradrenaline) will be necessary. A bronchodilator, such as aminophylline, may be useful in cases with severe dyspnea. The effectiveness of atropine and papaverine (vasodilator) is difficult to assess, although these drugs are widely used.

Anticoagulant therapy may reduce the chances of further embolization. If anticoagulants are used, I feel that treatment should begin immediately with heparin, the dose being controlled with clotting times. In addition, coumarin therapy is instituted. When the prothrombin time is sufficiently altered (about 30 seconds against a normal value of 13 or 14 seconds), heparin therapy is discontinued. Anticoagulation is usually carried on for 3-6 weeks, but may be continued indefinitely if indicated.

Finally, surgery has been employed to prevent further embolization — inferior vena cava ligation and unilateral or bilateral common femoral vein ligation have been used. The most successful treatment is, of course, prevention. Elastic stockings and leg exercises for immobilized patients and early postoperative ambulation will reduce the incidence of pulmonary embolus.

CARDIOGENIC SHOCK

Incidence:

Every practitioner of internal medicine will eventually be faced with the serious problem of cardiogenic shock. In a series of 714 patients with myocardial infarct studied by Heyer, 58 or about 8% developed shock. Other reports show the incidence of shock to be between 7 and 20%. Once severe shock has developed, the mortality is about 50%. Over one-half of the cases of shock develop within the first twenty-four hours and the majority developed within 72 hours. These figures indicate the need for particularly close supervision during the first 72 hours following the onset of the coronary occlusion.

Pathogenesis:

As in any type of shock, the pathophysiology and pathogenesis of the state is not completely known. In any shock state, normal blood pressure may be altered as a result of disturbance in one or more of three mechanisms—

1). cardiac output

2). tone of the peripheral vascular bed

3). an actual reduction in blood volume (*effective* blood volume is reduced in all shock states, as mentioned previously) in cardiogenic shock, a sharp drop in cardiac output because of the insult to the myocardium is the most important factor. The greater the infarction, the greater are the chances of shock developing. In response to the decreased cardiac output, there is peripheral vasoconstriction to maintain blood pressure. However, vasoconstriction physiologically is not maximum and can be enhanced by vasopressor drugs. If the physiological vasoconstriction is not sufficient to compensate for the decrease in cardiac output, then cardiogenic shock develops. In most instances, the clinical signs and symptoms of shock develop as the blood pressure falls to 80 or 90 mm. of mercury. However, in a few cases, transient hypotension without the clinical picture of shock may occur. The changes, if any, in blood volume are not known. Some cases of quick recovery following blood or plasma transfusion have been reported but most authors state that such therapy has no effect on the survival rate.

Clinical Aspects:

The clinical signs and symptoms are the same as for hypovolemic shock, with the added picture of myocardial infarction. There are three clinical types—

1). moderately severe, in which there is a prompt

response to vasopressors, which are discontinued in the first twenty-four hours — anuria does not develop.

2). severe, in which oliguria is frequent, and prolonged use of vasopressors is necessary. Digitalization is often required.

3). fatal, in which there is little response to vasopressor therapy with continued shock. Signs of heart failure usually develop, making digitalization necessary.

In about one-third of cases, there are one or more warning episodes of transitory shock with typical signs and symptoms and hypotension, which precede deep shock by one to several hours.

With prolonged shock, there is further insult to the myocardium with the additional possibilities of acute tubular necrosis of the kidneys, cerebral disorders, skin and muscle gangrene.

Treatment:

Prevention of shock may be accomplished by adequate treatment with analgesics, elevation of the foot of the bed, oxygen if there is premonitory shock and frequent blood pressure and heart rate readings in the first two or three days, so that therapy may be instituted promptly if shock occurs.

The only specific therapy is vasopressors, noradrenaline being the best. Eight mg. are placed in 1000 cc. of a 5% dextrose in water solution and given intravenously. Rate and amount of infusion is governed by the response.

Rapid digitalization is done in all cases of frank heart failure, and as a last resort in patients who do not respond to vasopressors.

Transfusions (intra-arterial or intravenous) of blood, plasma of I.V. solutions are not generally of any value.

Prognosis:

The prognosis depends on —

1). response to therapy — if a prompt response to vasopressors is obtained, with resumption of good urinary output, the prognosis is favorable. If anuria continues, the prognosis is poor.

2). rate of administration of vasopressors to produce a response is of prognostic significance.

3). duration of shock before therapy — most who receive prompt therapy within an hour of onset of shock will survive. Almost none survive if severe shock is untreated for 2½ hours or more. Additional factors which make for a less favorable prognosis are arrhythmias, heart failure and older age groups.

The relationship between the time of onset of therapy and the mortality rate indicates the great need for hospitalization of all myocardial infarcts and the taking of blood pressure and heart rate every hour for the first 2 or 3 days.

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(cont. from page 62)

facilities but a rather poor method of admitting patients. They were taken at first to the administration office like that of any government building, where a non-medical office employee assigned him to a particular ward; medical, surgical, obstetrical, etc. This explained why on rounds the professor several times apologized that a few patients were surgical cases who had been admitted to the medical ward because of the office's erroneous "diagnosis" and were waiting for the proper transfer papers to a surgical ward. The doctors were not unhappy but generally envious of Canadian doctors who were becoming wealthy because of their higher incomes.

Despite the minor differences in methods of diagnosis and therapy, I was interested to see that in the long run there is not much difference in results; I guess that is why each maintains its own preferences. But I feel very fortunate to have left my environment to see someone else's technique, and to realize that not only do they exist but that they are just as good.

B. A. Stein 6T4

Perforated Peptic Ulcer

D. Wilson 6T4

You are the junior interne on emergency. The following five patients are admitted to your service:

A). A 48 year old woman with a ten year history of radiologically-proven duodenal ulcer who 2 hours ago developed sudden agonizing epigastric pain extending to both shoulders. On examination there is board-like rigidity, tenderness, abdominal distention, temperature 103°, pulse rate 125, blood pressure 80/40, and respiratory rate 44. Abdominal x-ray shows air under the right diaphragm.

B). A 48 year old woman who 2 hours ago developed sudden severe epigastric pain referred to the right flank. On examination there is a board-like belly, abdominal distention, tenderness, temperature 102°, pulse rate 110, blood pressure 125/85, respiratory rate 44. Abdominal x-ray is normal.

C). A 24 year old man who 10 hours ago began to complain of pain and tenderness in the right lower quadrant, associated with anorexia, nausea, and vomiting. Physical examination reveals a rigid abdomen, diffuse tenderness, absent bowel sounds, wbc 12,000, temperature 99.8°. Abdominal x-ray is normal.

D). A 30 year old, shocked alcoholic who began to feel pain 4 hours ago, epigastric in location, radiating to the back, becoming very severe, and associated with vomiting. The abdomen is distended, rigid, tender, and shows rebound tenderness to the epigastrium. Heart rate is 130, temperature 102. Serum amylase is 300. Abdominal x-ray is normal.

E). A 50 year old female who has had mild epigastric pain for 3 months. She came to the hospital because her pain suddenly became severe and she vomitted profusely. On examination she appears shocked, her abdomen is rigid, bowel sounds are diminished, heart rate is 120, respiratory rate 40, and blood pressure is 80 systolic.

Have you made your diagnoses?

A is a classical case of perforated peptic ulcer.

B is suffering from acute cholecystitis.

C has perforated an ulcer he never knew he had. The fluid is draining down into the right iliac fossa.

D Don't let the serum amlase fool you. D has perforated a duodenal ulcer, at the same time inflaming his pancreas.

E Better do an ECG on this woman who has amyocardial infarction.

As a matter of fact, any of these patients could have been diagnosed as perforated ulcer by an experienced surgeon. A was treated by simple closure, B had a cholecystectomy after the stomach and duodenum were extensively explored for a leak, C had a healthy appendix removed before his perforation was sewn up, D was treated conservatively and died the next day with a fulminating bacterial peritoneal infection, E fortunately did have an ECG and was successfully treated for myocardial infarction.

The perforation of a peptic ulcer is a surgical emergency which requires knowledge, insight, experience and luck for a diagnosis. Despite low mortality rates reported in various selected series, the average mortality rate in large general hospitals ranges from 10% to 20%.

Etiology

The actual cause of the perforation is still a matter of conjecture. Of 514 cases in one series,[2] 81 patients were drinking at the time of the perforation. Many were eating shortly before. Emotional stresses seem related in some cases. In 2 cases, it occured while the patients were having sexual relations for the first time.

Perforation is more likely to occur in acute ulcers with a very short history in which there is rapid penetration of the muscle wall. In chronic ulcers there are likely to be adhesions. Ulcers with continuous symptoms are more dangerous than those with intermissions for in the former there is no healing and little fibrosis. The long-continued administration of cortisone has increased the danger of perforation. Prednisone, triamcinolone and PAS are also thought to be responsible. It is not unusual following massive x-ray therapy (5000 r) when the stomach is in the radiation field.

Physicians should inform their patients with an ulcer of the dangers leading to perforation; the possible effects of excess alcohol ingestion, the necessity to avoid emotional stress, and the need to seek help whenever abdominal pain is steady and not relieved by the usual anti-pain methods.

Site

About 88 per cent of perforated ulcers are duodenal. Ninety-eight per cent of these being in the anterior part

of the superior portion, the remainder in the descending duo-denum. Ten per cent are stomach in position of which 40 are prepyloric, 30 antral, 25 midportion and 5% cardia. Two per cent are marginal and jejunal.

Diagnosis

In 80% of cases, the history reveals that the patients have had symptoms suggestive of peptic ulcer or that roentgenologic examination had proven the presence of an ulcer. However, as evidenced by the 5 cases presented, the diagnosis is often difficult.

The diagnosis is usually obvious when manifested by sudden pain in the upper abdomen with tenderness and muscle rigidity, diminished or absent bowel sounds, obliteration of liver dullness and demonstration of pneumoperitoneum on x-ray study. In support of the diagnosis there is usually a temperature greater than 102°F., pulse rate more than 120, respiratory rate of at least 40, and blood pressure less than 80. There may also be associated hemorrhage and pulmonary rales.

The pneumoperitoneum is usually visualized under the right hemidiaphragm and may be enhanced if the patient sits upright for 20 minutes before the x-ray examination or if the right lateral decubitus position is used. In the absence of subphrenic air (47 per cent of cases), the diagnosis may be more difficult.

When one can rule out pneumonia and coronary thrombosis, the majority of diseases producing this symptom complex are intra abdominal processes that require surgical intervention — that is, acute cholecystitis, appendicitis and perforated diverticulitis. The one condition that has given the most difficulty is pancreatitis which does not require operation. Even here, the problem is not so much the pancreatitis which is explored, as it is the perforated ulcer mistaken for pancreatitis and not explored. As seen in case D, the chief difficulty is the high serum amylase activity which may be associated with perforated ulcer and lead to the incorrect diagnosis of pancreatitis. A patient with the signs that D presented can often be safely handled with gastric suction and antibiotics since the most likely disease is edematous pancreatitis or a self-sealing ulcer.

The diagnosis is most difficult in cases in which the patients are not observed until 24 to 48 hours or more after the perforation. In such cases, some localization of peritonitis has occurred and bizarre intraperitoneal abscesses often obscure the true nature of the lesion.

The most frequent erroneous diagnosis is acute appendicitis as in C or acute cholecystitis.

Mortality

Mortality rates are very low during the first 6 hours, but increase with every 6 hour interval. After 24 hours, the rate is between 40 and 50 per cent.[4] This mortality is due to extensive fluid loss into the abdominal cavity secondary peritoneal bacterial infection and a weakened cardiopulmonary reserve of the patient.

The infecting organisms are Staph, aureus, hemolytic strep-alpha and beta, strep-fecalis and candida in that order.

Treatment

There are 3 different approaches by surgeons today. These include: (1) conservative therapy with nasogastric suction and fluid and electrolyte replacement; (2) radical partial gastrectomy at the time of perforation; (3) simple closure of the defect with interrupted sutures and an omental tab. Toronto surgeons are generally in favor of and employ the third regimen.

Basically, the admitting interne's responsibilities are to establish the diagnosis, to insert a Levin tube through the nose and begin gastric suction, to administer fluids and blood IV to correct dehydration and to combat shock and anemia, and to prescribe antibiotics for infection. In most cases, operation is advisable within 12 hours after the onset of symptoms. When the clinical situation is doubtful, it is better to explore such cases than to permit serious abdominal disease to progress unchecked.

The patient who enters at a long interval after perforation may have a low blood pressure and blood volume. Surgery should not be performed until adequate fluid, electrolytes, plasma and blood replacement have returned the pressure and urine flow to normal. Broad spectrum antibiotics and nasogastric suction should be instituted.

1. *Conservative therapy*

Constant medical care of suction and fluids has been reported in selected series to give lower mortality figures than simple suturing of the perforation.[2] It is based on the assumption that the perforation is usually sealed at the time of surgery. However, these figures are deceptive: they usually exclude the patients who are moribund on admission, they are younger patients, the diagnosis is not always certain, and most are seen early after perforation.

2. *Radical surgery*

Elective subtotal gastrectomy at the time of perforation is becoming popular in some centres. This is in contradistinction to a mandatory gastrectomy as with discovery of cancer, severe bleeding, obstruction, or very large perforations that would be dangerous to close.

Gastrectomy can be performed with a low mortality and morbidity rate when there is minimal peritoneal contamination, relatively healthy first portion of duodenum, general good health, a competent anesthetist and blood bank, and a trained team for gastric surgery.[2]

The procedure has been slow in acceptance since only a third of the patients with perforated ulcer requires subsequent gastric surgery. However, half of the remainder have continued ulcer complaints.

There are surgeons who accept this approach when qualified by certain requirements; if there are indications of a severe ulcer diathesis — previous perforation, hemorrhage or obstruction, a long history of ulcer distress associated with back pain, gastric ulcer, perforated jejunal ulcer, perforation of duodenal ulcer if the ulcer is obviously chronic at time of operation, and multiple ulcers, it appears reasonable to perform definitive ulcer surgery at the time of perforation in a good risk patient.

3. *Simple closure*

The usual surgical treatment of the perforation consists of simple closure of the defect with the use of (1) interrupted sutures and an omental tab or (2) excision of the ulcer and suture, or (3) cauterization of the ulcer and subsequent suture. Toronto surgeons generally use the first procedure almost routinely when the diagnosis is established. A local study 12 years ago revealed that about a third are cured by this operation, a third are well handled medically following closure, and the remaining third eventually required definite surgery. The argument against radical surgery is — why resect part of every patient's stomach when only one third will actually require it? Why expose all patients to the possibility of a dumping syndrome

Ideally, laparotomy is deferred until the patient's initial prostration has abated. In addition to the essential diagnostic studies, preparation of the patient includes the insertion of an indwelling gastric catheter and intravenous infusions to restore a normal blood volume and to correct losses of fluids and electrolytes incurred by vomiting. The chief virtue of the closure method lies in its simplicity and in the established fact that it is an effective remedy for the emergency condition and that it permits diagnostic accuracy. Closure can be performed during a relatively short anesthesia; local anesthesia may suffice. For this reason, closure often imposes the least burden on the patient.

Post operative management should include as strict a medical regimen as for any open active peptic ulcer. This includes diet, antacids, anticholinergics, sedatives, avoidance of cigarette smoking and alcohol ingestion, and psychotherapy where indicated.

In patients, in whom a diagnosis cannot be absolute, it seems safer to explore and be certain than to wait until peritoneal infection has developed. This procedure also rules out gastric ulcer with its higher mortality rate and danger of malignant change.

However, in patients who arrive at the hospital more than 24 hours after perforation with minimal tenderness in the epigastrium, no signs of free abdominal fluid and no increase in pulse rate or temperature, the perforation has probably sealed and can be treated conservatively. Patients who are moribund on admission and who do not respond to the measures previously outlined have to be treated conservatively. The same limitation probably holds true for patients with severe cardio pulmonary disease.

Results of Treatment

Doctor Tovee's results locally have been mentioned. The following includes reports from many different centres:

METHOD OF TREATMENT

	Closure			Resection			Nonoperative			Overall	
	Cases	Deaths	%	Cases	Deaths	%	Cases	Deaths	%	Cases	Mortality %
1940-46	9,381	2,259	24.1	3,612	497	13.7	—	—	—	12,933	21.2
1950	10,567	1,186	11.2	4,954	340	6.9	2,085	240	11.5	17,606	10.0
1959	19,948	3,445	17.3	8,566	837	9.7	2,085	240	11.5	30,599	14.8

Here, the group treated by simple closure probably represents the widest spectrum of cases and the least selection. By contrast, the group treated by immediate resection probably represents the greatest descrimination. A considerable proportion of deaths in all categories result from factors inherent in the perforation and irrespective of the method employed. The commonest recorded cause of death is peritonitis, almost invariably induced by the perforation and not by the treatment. It is apparent that the overall immediate mortality rate in reports since 1950 has been reduced to slightly less than half that recorded in 1940 and 1946.

Mortality is adversely affected by delay in treatment, widespread peritineal contamination, chronicity in the ulcer, and debility in the patient. Mortality is comparitively higher in elderly patients, in females, and in gastric ulcer. Generally there is almost equal mortality following laparotomy with simple closure and after nonoperative treatment.

Long term follow-up after acute perforation in 4542 collected cases reveals these results:

It appears that the sequelae are similar.

The preponderant indication for definitive surgery following perforation treated by closure is obstruction. Hemorrhage occurs in about one-fourth of cases. A second acute free perforation occurs in about 5 per cent.

ACKNOWLEDGEMENTS

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Method of Treatment	Symptom-free			Recurrent Ulcer Symptoms			
				Treated Medically		Requiring Surgery	
	Total Cases	Cases	%	Cases	%	Cases	%
Closure	3974	1186	29.8	1357	34.2	1431	36.0
Nonoperative	290	102	35.2	80	27.6	108	37.2
Resection	278	223	80.2	52	18.7	3	1.1

(cont. from page 66)

Cardiac resuscitation is outlined and the emergency procedures involved are emphasized. Although myocardial infarction is the commonest emergency it is well to remember that serious arrhythmias are usually the terminal event not only in this but in almost all deaths of a cardiac origin.

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Recent Advances

MEASLES VACCINE

Whenever a physician inquires into the past health of a patient, he asks specifically regarding such maladies as rheumatic fever and scarlet fever but passes off (as does the patient himself) such "inconsequential" diseases as mumps, measles, and chickenpox. These are the almost inevitable, inconsequential diseases that every child contracts. This disregard does indeed have a sound basis, for statistically the mortality and morbidity of the diseases are very low (for example, measles causes 0.2 deaths/100,000 population). Yet in the past few years a vaccine has been developed against one of these disease entities — measles.

The virus of Rubeola was first isolated in 1954 by Enders and Peebles in Boston, paving the way to the development of a vaccine. The first field trials on such a vaccine were completed in 1959, and there is still a large amount of investigation being devoted to the study of the antigenic potentiality, the clinical ifficiency, and the occurrence of side reactions. There are four different methods by which the vaccine may be administered:

(1) Live attenuated vaccine (LV);

(2) Live attenuated vaccine plus measles immune globulin which is given in different site (LV + IG);

(3) Killed vaccine (KV);

(4) Killed vaccine followed by live attenuated vaccine (KV + LV).

It has been found that LV is very effective, producing a sustained a/b response. However, it induces fever (of 103° F. or greater) and rash in a significant number of children (30-40%), beginning about the sixth day and lasting two to five days. LV + IG give a reduced number of reaction (only 15%) and those with reactions have a shortaer course. Both of these forms have a 95% a/b response, with a duration of protection of at least two years and a steady persistence of a/b for at least three years. The KV gives protection for at least several months, and serological conversion after three monthly doses is approximately 90%. The only side reactions encountered are minor and similar to those of other alum-precipitated vaccines. However, a/b titres are transient and usually disappear within a year, thus requiring a booster dose. The combination, KV + LV, with the live administered one month after one to two doses of killed, gives a/b titres comparable to LV alone, with a marked decrease in fever and rash (10% side effects). Studies show that effective immunity persists for at least six months. In the light of the fact that KV a/b levels are transient (and, thus, probably the immunity as well), its use is usually limited to groups in which the use of live attenuated vaccine is contraindicated, such as in pregnancy, in leukemia, lymphomas, and other generalized malignancies, and in marked egg sensitivity. LV + IG or KV + LV seem to be the vaccines of choice, and since the latter is associated with somewhat fewer side effects, it will probably become the more popular.

These statistics speak well for the efficacy of the measles vaccine, as well as for its safety. Thus, the possibility is good that this vaccine may become part of the regular immunization schedule, along with those of diphtheria, pertussis, tetanus, and polio. (Indeed, a combined measles-polio vaccine has already been field-tested, using inactivated measles and polio viruses, and has been found to give as high a/b titres in combination as each vaccine used separately.) The only problem seems to be, as stated in the introduction, that all this development serves only to eliminate an inconsequential disease.

However, despite popular attitudes, measles is not always a benign disease that can be lightly disregarded. A certain number of children contracting measles develop an associated pneumonia (due to the measles virus) which may lead to acute pulmonary insufficiency and death. Approximately one in every thousand has as a complication, measles encephalitis (probably due to a hypersensitivity reaction). 10-30% of such cases die, while 40% of the survivors show permanent sequelae of mental retardation, personality changes, and behavioral disorders. Even in its "benign" course a child with measles undergoes a three to four day period of acute illness with high fever, severe cough, headache, and itching and burning of the eyes. This latter aspect of the disease would itself warrant prophylaxis and the serious complications add further justification.

Thus, the effort to develop an effective measles vaccine seems well worthwhile, and it would seem to be only a matter of time before measles becomes a disease which is not inevitable but, rather, unusual. B.K.

Answers to On Inspection

C. Listgarten 6T4
G. Harper 6T4

FIGURE 1: Intussusception a. whole specimen, b. longitudinal section.

This is a case of jejunal intussusception occurring in a 61 year old woman. One hour after eating turnips, she noted severe crampy epigastric pain radiating to the back and relieved by repeated whitish, blood- and bile-free vomiting occurring with the intake of all foods and liquids. There was never any melaena. Examination revealed a firm distended abdomen without mass or localized tenderness.

Operation three days later for suspected intestinal obstruction uncovered a Richter's left femoral hernia which was repaired. Postoperatively the patient's condition worsened and she died on the fourth postoperative day. Autopsy disclosed this jejunal intussusception. Acute intussusception is rare in adults and nearly all cases are secondary to a pathological lesion such as a polyp, lipoma, etc . . . Occasionally it follows the ingestion of a foreign (Non-digestible) body, or abdominal injury with the formation of a submucosal hematoma.

Treatment is always by resection in adults, since malignancies form the leading point in 50% of cases.

FIGURE 2: Electrical burns a. pre-operatively, b. post-operatively.

This 43 year old hydro worker grasped a 7200 volt line with his left hand and held it against his abdomen. At Operation five days later, the left arm was amputated and a pedicle graft was swung over to cover the defect in the debrided right upper quadrant. Ten days after surgery he suddenly discharged large amounts of old dark brown blood and yellow fluid from his abdominal incision. At operation a fistula of the small bowel was found and repaired by an end-to-side ileocolostomy.

Electric burns are of two types:

(1) Flash burns — due to the heat released.

(2) Current burn — due to the passage of current through the body.

The latter are more common and more severe (usually third degree), and may be accompanied by flash burns of lesser degree. The points of entrance and exit of the current are marked by whitish well defined areas, surrounded by hyperemia, where the tissue is often cooked down to the bone. If a part is held against the current source, it may become charred or mummified. Edema follows the initial injury and necrosis extends well beyond the burn area due to vascular thrombosis.

Sequestration of dead tissue is slow and healing is delayed, the ulcer being unreceptive to grafts. Excision of dead tissues should be delayed for a week as full necrosis is not often apparent till then.

FIGURE 3: The Cardioverter in use at St. Michael's Hospital, Toronto.

The Cardioverter is the most recent method developed for the treatment of cardiac arrhythmias. Lown et al., who developed it, describe the apparatus as consisting of four components:

(1) A direct current depolarizer which supplies the charge.

(2) A cardiometer which follows the heart rate and provides an R wave as baseline for the synchronizer.

(3) A synchronizer which delivers the charge at any desired time in the cardia cycle.

(4) A cardioscope which supplies continuous E.C.G. monitoring.

The patient is lightly anaesthetised with I.V. Thiopental. An impulse of 2.5 msec. is delivered to the closed chest. The synchronizer is set so that the impulse is delivered 20 msec. after the peak of the R wave, thus avoiding the vulnerable period on the upstroke of the T wave. This preselection totally eliminates the risk of ventricular fibrillation. At first a 100 watt-sec. discharge is given. If it fails to reestablish normal rhythm, the energy dose is increased by 100 watt-sec. steps up to a maximum of 400 watt-sec.

Each charge is accompanied by a muscular twitch and an audible sigh. The total procedure takes less than half an hour and the patient is conscious in five minutes. The patient is then placed on maintenance doses of quinidine to prevent recurrence of the arrhythmia.

The advantages of the Cardioverter as compared with the time-honored use of quinidine are, according to Lown et al., that:

(1) It is simple, quick, and safe.

(2) It is effective in 90% of cases; quinidine in 50%.

(refer back to page 70)

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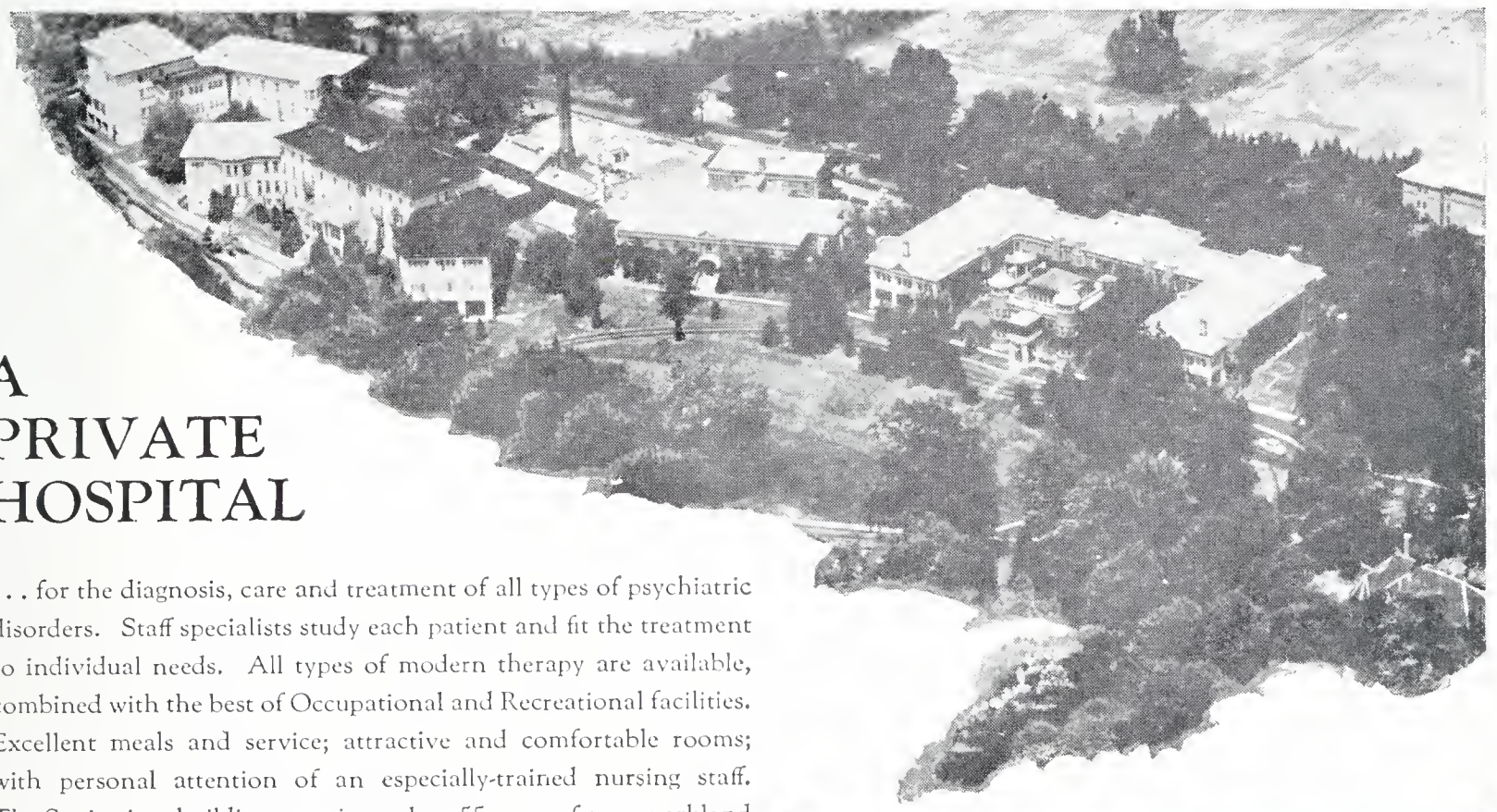

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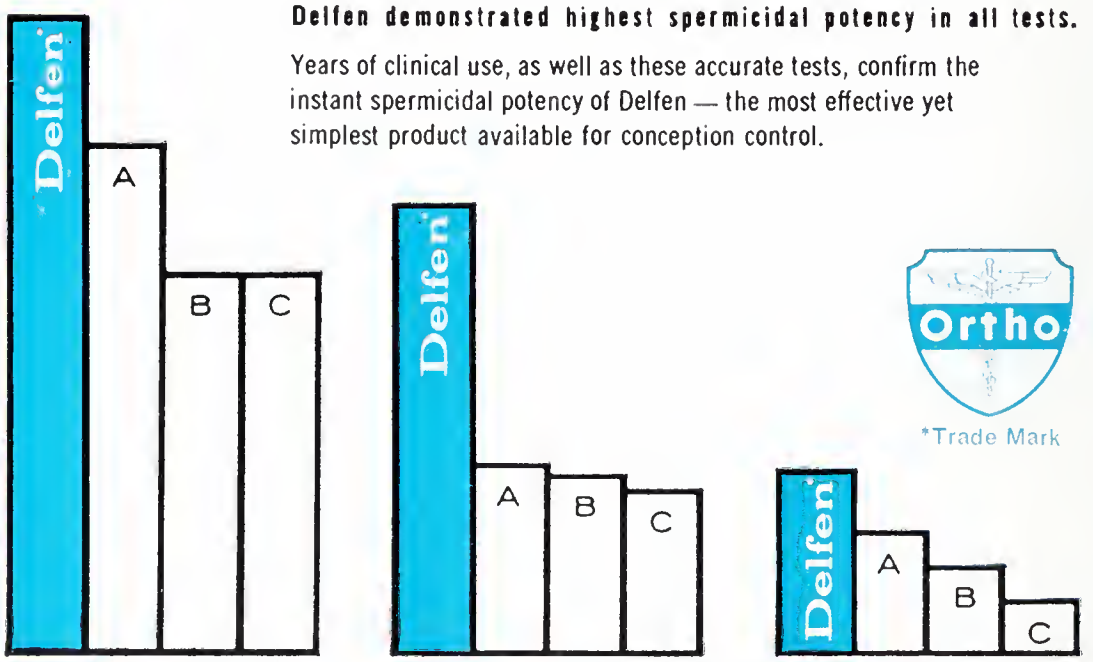

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